SUNSTROKE

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THERMIC FEVER,

OR

SUNSTROKE.

BY

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BOYLSTON PRIZE ESSAY.

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TO

DR. S. WEIR MITCHELL,

Not only as a tribute to his high renown as an original investigator, and an offering of personal attachment; but also as a grateful acknowledgment of practical sympathy and generous aid to the author in his early attempts at medical research,

THIS BOOK IS RESPECTFULLY

Dedicated.

PREFACE.

About ten years ago my attention was directed very forcibly to the subject of sunstroke by the cases which I witnessed at the Pennsylvania Hospital, when resident physician. At that time I noted the peculiar rigidity of the heart after death, about which so much is said in this essay. The great interest excited in me by the disease, and the fact that my observations were rather derided in certain quarters, long since determined me to make at some time an experimental study of the subject. The present essay is the result of this determination.

Although my effort has been awarded the Boylston Prize of the Harvard University, yet with some hesitation I place it before the medical public, claiming only that I have endeavored simply to find the truth, and asking that any

shortcomings may be pardoned, because my work has been honest in its intentions and desires.

The following is extracted from the minutes of the Boylston Medical Committee:

"By an order adopted in 1826, the Secretary was directed to publish annually the following votes:

"1st. That the Board do not consider themselves as approving the doctrines contained in any of the dissertations to which premiums may be adjudged.

"2d. That in case of publication of a successful dissertation, the author be considered as bound to print the above vote in connection therewith."

PART I.

CLINICAL HISTORY.

Introductory.—There can be no doubt that under the name of sunstroke or *coup de soleil* sudden cases of severe illness of very different natures have been described by authors. Such of these cases as have really been dependent upon exposure to excessive heat can be classified under two, or perhaps three, heads, to which the names of *acute meningitis* or *phrenitis*, *heat exhaustion*, and *thermic fever* or *true sunstroke*, may be respectively applied, as more or less expressive of the pathological conditions existing.

Acute meningitis or phrenitis, due to exposure to the sun and the direct action of its rays upon the head, must be a very rare affection. In fact, I have no positive evidence to offer of its existence in nature, having never seen or read an unequivocal record of such a case, and therefore will pass this theoretical class by without further allusion.

Simple exhaustion, due to excessive labor in a

heated atmosphere, is an affection so very distinct from true sunstroke, that it is strange it should ever have been confounded with the latter. It does not differ in its pathology or symptoms from other forms of acute exhaustion, offering like them, as its chief features, a cool, moist skin, and a rapid, feeble pulse, associated with great muscular weakness, and a tendency to syncope. The following case is the only one I have seen; the notes are from memory, and therefore not so full as might be desired.

CASE. ——, æt. about 50, was brought into my office at 1 P.M. of a hot July day. He appeared to be a man of feeble physical organization, and, after rallying, stated that he had been feeling exceedingly weak and ill for several days. He was in a semi-unconscious, syncopal condition, with an exceedingly rapid, fluttering pulse, a cool, damp skin, and pale face. I had him laid flat upon his back, and brandy administered very freely. Under its use the pulse soon became much steadier and slower; half an hour afterwards he was able to walk with assistance to a carriage; and I heard of him no more.

Some years ago the late Prof. Wm. Pepper (American Journ. Med. Sci., Jan. 1851) reported to the College of Physicians certain cases of sunstroke, in which on post-mortem examination he found the heart very much softened and relaxed. As he distinctly states that the surface during life was cool, there can be but little doubt that his

cases really belonged to the affection now under consideration.

In the discussion that followed the reading of his paper, Dr. Condie described very clearly the two classes of sunstroke cases, and was, so far as I know, the first American writer who clearly recognized the distinction. He evidently believed Dr. Pepper's patients to be suffering simply from exhaustion.

As there is nothing peculiar in these cases, I do not think that they should have any especial name. The term heat-exhaustion might be applied to them had it not been used to signify true sunstroke. The main point to be borne in mind is, however, that such cases should not be called sunstroke, as they have not the slightest affinity with that disorder.

Causation.—It is the third class of cases, then, to which I would restrict the name of sunstroke, and which alone offer a disease *sui generis*, as well marked and distinctive in its clinical history and anatomical lesions as any affection at present known.

Although this paper has for its chief object the discussion of the pathology of sunstroke, yet a sketch of the clinical history is an evident necessity to its completeness.

In the first place, in regard to the etiology of the disease, my own experience is, that the only abso-

lutely necessary, and the ever-present, immediate cause, is heat, solar or artificial. It was formerly believed that exposure of the head to the direct rays of the sun was requisite, but this is now well known not to be true. One of my own cases originated in a sugar refinery.

Dr. Longmore tells us that out of sixteen cases seen by him in one epidemic, thirteen originated in barracks or hospital.

M. Boudin, as quoted by Dr. W. C. Maclean (Reynolds's Syst. of Med., vol. ii. p. 157), states that one hundred cases of sunstroke occurred on the French man-of-war Duquesne, most of them at night, when the men were lying in their bunks.

Dr. Morehead (Researches on Diseases of India, vol. ii. p. 583) says that direct exposure to the sun, although a frequent, is not a necessary condition to the production of the disease.

Dr. Geddes (Clinical Observations on Diseases of India, London, 1846) details a case which occurred in the hospital in the person of a man convalescent from measles.

Dr. Barclay says (Madras Quarterly Journal, No. 2, 1860), "The attacks of insolation came on, with very few exceptions, when the men were in the tents,—generally during the day, but in several instances during the night,—never, except in one instance, along the line of march. The patient had generally been lying down, and often seem-

ingly asleep, or, as it would probably be more accurate to say, attempting to induce sleep. Sometimes the attention of his comrades was first directed to him by his hurried and heavy breathing, and on attempting to arouse him he was found insensible."

Dr. Bonnyman (Edinburgh Med. Journ., vol. xiv. p. 1029, 1864) says, "By far the greater number of cases that yearly occur in India are of men who have not been exposed to the sun. It is not unusual for men to go to bed in apparent health, and to be seized during the night; and patients in hospital, who have been confined to bed for days previously, are frequently the subjects of attack."

Dr. Swift (p. 47) asserts that the same affection may be caused by exposure to artificial heat, and that eleven of his cases were attacked in the laundry of a hotel, and several were brought from sugar refineries.

These quotations could be very much increased in number, but they are surely sufficient to show that exposure to the direct rays of the sun is in no degree necessary for the production of *coup de soleil.* The testimony of authors, and that of common experience, that sunstroke occurs only in a highly-heated atmosphere, are indeed so concordant, that it is certainly allowable to take this point for granted as long ago proven.

There has, however, prevailed in certain quarters an idea that intemperance, and even malaria, were

necessary factors in the production of *coup de soleil.*

There can be no doubt that in this country a large number of the cases occur in the persons of habitual drinkers; but the most temperate are not exempt.

In India, among the European soldiery, the temperate would seem at times to afford even the larger proportion of victims, because the vagabond drunkard soldiers are notoriously shirkers of duty. Thus, of eight fatal cases in the 71st Highlanders, occurring on the 7th of May, 1858, six were sober men, one a "tolerably free liver," and one a "free liver" (Crawford, Madras Quarterly, No. 2, p. 30, 1860). Dr. Crawford also says (p. 315), "I can point to many soldiers whose lives are spent in an unvarying round between the canteen, the guard-room, and the prison, and yet they do not die of insolation at a time when our best non-commissioned officers are victims to the apoplectic form of ardent fever." Both he and Dr. Barclay also insist that malarial poison is in most instances of insolation not present, and does not when existent strongly predispose to attacks.

The real and sole producing cause of the disease is, therefore, *Heat.* There are, however, various predisposing causes, which greatly aid the exciting cause probably by lessening the powers of resistance to it. First in importance among these is a

want of acclimatization. Foreigners are always attacked in much larger numbers than natives of the tropics. It must be remembered, however, that no amount of acclimatization will afford certain protection, as even the Hindoo, born and bred in the stifling atmosphere of Bengal, is occasionally attacked.

A second important predisposing cause is habitual intemperance, all or almost all authorities coinciding in the assertion that habitual drinkers afford a much larger proportion of victims than temperate men when the exposure is the same.

A third predisposing cause is debility, and especially that debility or exhaustion which has been brought on by fatigue, and in the highest degree by fatigue in a heated atmosphere. The most frightful epidemics on record have had this predisposing cause for their ground-work.

A very marked instance of such an epidemic is that recorded by Dr. Barclay (loc. cit.). The 43d Regiment, to which he was attached, made a most extraordinary march of over eleven hundred miles, chiefly through the lowlands of India, and at the hottest season of the year. This march was continuous, with the exception of a few brief halts. No cases of sunstroke occurred until nine hundred and sixty-nine miles had been traversed, and the men had become thoroughly exhausted, and even markedly emaciated. Shortly after this

the regiment rested some eight days, and then started again, arriving soon in a valley in the Bisramgunge Ghat, a narrow ravine, with precipitous walls nearly a mile in height. During the day the thermometer in the tents ranged from 115° to 127°, and on one occasion was noted 105° at midnight. The number of cases of insolation now became very great, and although most of them recovered, two officers and eleven men were lost in the four days during which the regiment remained encamped. Although the air became cooler as the regiment emerged from the hills, yet seven more fatal cases occurred in three days. The further following of Dr. Barclay's account is not necessary for our purpose. It certainly shows that exhaustion from severe labor at a high temperature is a powerful predisposing cause of sunstroke.

Another predisposing cause, according to the testimony of Indian observers (especially Dr. Barclay, p. 378), is a febrile state. Some have deemed that malaria was especially active in this respect, but, as Dr. Barclay states (loc. cit., p. 378), "at the season when insolation prevails, malaria is never present in the air in any quantity."

Still another predisposing cause appears to be crowding in badly-ventilated barracks. Dr. Butler says, as quoted by Maclean, "Assuredly those barracks the most crowded, least ventilated, and worst provided with punkahs and other appliances

to moderate excessive heat, furnished the greatest number of fatal cases." Mr. Longmore says, "Nearly half the deaths occurred in a single company of the regiment quartered in the barrack which was manifestly the worst conditioned as to ventilation, and, indeed, in every sanitary requirement."

A predisposing cause, which in the past has added to the mortality list of sunstroke among British soldiery, has been improper clothing. "The buttoned-up, tight-fitting coats, the leather stocks, as stiff and unyielding as horse-collars, the heavy cross-belts, so contrived as to interfere with every movement of the chest, the heavy felt shakoes mounted with brass ornaments, with wide, flat, circular tops, ingeniously contrived to concentrate the sun's rays upon the top of the head," no doubt very seriously interfered with the soldier's power of resisting the high temperature.

In reviewing these various predisposing causes, it will be found that all of them either render the nervous system more sensitive to the morbific agent,—heat,—or else interfere with the glandular system, so weakening it that it is unable to afford the proper secretions whose evaporation shall enable the body to resist the external heat, or perhaps act in both of these ways.

Thus, want of acclimatization means simply an unaccustomed condition of the nervous system to the stimulant, heat, and also, probably, that the

glandular system, from want of training, so to speak, is unable readily to produce the requisite amount of secretion. Again, intemperance causes the same results in a slightly different form: organically weakened nervous system and organically altered glandular apparatus are unable to cope with the morbific agent.

Symptoms—Course.—The idea that sunstroke attacks its victims instantaneously certainly obtains among the people, and to some extent even among the profession.

The truth seems to be, however, that in most instances the affection gives abundant warning of its approach, although in others it attacks very suddenly. The histories of nearly all the cases herein reported were very imperfect, yet in one (Case 5) the wife of the sufferer gave a very distinct statement that the man had had severe headache, vomiting, and weakness previously to the attack. An examination of clinical records shows much more positively that prodromes, or symptoms preceding unconsciousness, are very common, and, indeed, generally present.

Dr. Morehead, in his work on the Diseases of India, clearly recognizes their existence. He says, p. 583, "In this degree there is much headache, with intolerance of light, and contracted pupils, succeeded by suffused eyes and drowsiness or quiet,

delirium, or convulsions,—followed by coma with dilated pupils."

Dr. Longmore (London Lancet, March, 1859) calls attention to excessive irritability of the bladder as a symptom preceding attacks of sunstroke.

Dr. Bonnyman (loc. cit.) says, "Where premonitory symptoms show themselves, they are sometimes well marked. Those usually observed are, —inaptitude and disinclination for any exertion, drowsiness, or a desire to sleep, vertigo, headache, and slight confusion of ideas; the patient feels weak, sighing frequently; the appetite is gone, thirst is increased, and the bowels are constipated; the symptoms become aggravated, and the patient either passes into the state of profound coma, or symptoms of the first or progressive form of the malady are complained of, viz., distressing headache, with a feeling of weight and heat in the occiput, tightness, distention, and throbbing in the forehead and temples, anxiety at the præcordia, nausea, and a disposition to vomit. A sensation of sinking or of insupportable weight, or uneasiness, is referred to the pit of the stomach, and a feeling of horror or of impending calamity, with a tendency to weep, is experienced. The breathing is natural, or slow and sighing. The face is generally natural or somewhat flushed, eyes bright, pupils either natural or somewhat contracted. The skin is very hot and dry; the pulse is full and

accelerated, tongue white, thirst intense, bowels confined, the *urine suppressed.* If these symptoms persist, tetanic convulsions suddenly appear, and the patient lapses into the second or severe form of the disease."

Dr. J. R. Taylor says ("On Erethismus Tropicus," London Lancet, 1858, vol. ii. p. 355), "The symptoms in these cases were, weakness, pains in the limbs, great oppression at the præcordia, and, to use the patient's own words, 'a burning pain inside.' An indefinite pain in the head was always admitted upon inquiry, and sometimes, but not in many instances, was the most prominent cause of complaint."

Dr. Swift states that premonitory symptoms were wanting in the majority of his cases, but that the insensibility was very generally preceded by pain in the head, disordered vision, a sense of weight in the epigastrium, and sometimes nausea and vomiting. Surrounding objects appeared of a uniform color, in most cases blue or purple, in some red, green, or even white.

Dr. Staples (British Army Reports, 1868) states that as seen by himself prodromes were always present, the disease appearing in fact to come on gradually, there often seeming to be no line of demarkation between the prodromatous stage and the stage of complete insensibility, the latter coming on gradually.

All the cases of sunstroke which have come under my observation have been in the hospital, and therefore represent only the severe, fully-formed disease. The symptoms have been very constant. Total insensibility was always present, with, in rare instances, delirium of the talkative form, and still more rarely the capability of being roused by shaking or shouting. The breathing was always affected, sometimes rapid, sometimes deep and labored, often stertorous, and not rarely accompanied by the rattle of mucus in the trachea. The face was often suffused, sometimes, with the whole surface, deeply cyanosed. The conjunctiva was often injected, the pupils various, sometimes dilated, sometimes nearly normal, sometimes contracted. The skin was always intensely hot, and generally, but not always, dry; when not dry, it was bathed in a profuse perspiration. The intense burning heat of the skin, both as felt by the hand and measured by the thermometer, was one of the most marked features of the cases. The degree of heat reached during life was, in my cases, mostly 108°–109° F. The pulse was always exceedingly rapid, and early in the disease often not wanting in force and volume; later it became irregular, intermittent, and thready. The motor nervous system was profoundly affected: *subsultus tendinum* was a very common symptom, great restlessness was also very often present, and sometimes

partial spasms or even violent general convulsions. The latter were at times epileptiform, occurring spontaneously, or they were tetanoid and excited by the slightest irritation. Sometimes the spinal cord appeared to be paralyzed, the patient absolutely not moving.

Dr. Barclay also notes this difference in cases in regard to the motor apparatus. He says (p. 364), "In a large proportion of cases, from the commencement of the attack till its termination in death the patient never moved a limb or even an eyelid. A comparatively small number of cases, however, were from the first attended with convulsions. These generally began in the upper extremities, and in some cases they did not extend farther, the patient either becoming rapidly insensible or recovering; but in other instances they extended to the whole of the voluntary muscles, and were of the most violent description,—ceasing frequently for from two to three, to fifteen or twenty minutes, and recurring again with increased severity."

Petechiæ and ecchymosis, the evidences of broken-down blood, were present in some of my cases, and there was, in one or two instances, even a fetid hemorrhagic exudation from the nostrils during life. A symptom which has pretty much escaped the attention of authors was the peculiar odor. This was most marked in those patients

who had involuntary passages, but was very distinct from any fecal odor. The stools emitted it very strongly, but so did the skin and breath. It was so distinctive as to render possible the recognition of a case by the sense of smell alone. The discharges from the bowels were liquid and very often involuntary. None of the cases passed urine whilst under observation.

These symptoms agree very well with the descriptions of most authors. Dr. Barclay (loc. cit.), however, says that in his cases "the eyes were fixed and slightly turned upwards, becoming gradually more and more glassy, as if from the formation of a film over the cornea; the pupils greatly contracted; the conjunctiva pinky, the color gradually becoming deeper; the congestion at first being deep-seated. The face was invariably pale, never in any instance bloated or flushed, as has been described by others. The heart's action was very rapid and sharp, the impulse and the pulsations in the carotid being perceptible to the eye from a considerable distance."

The truth seems to be that all the minor symptoms vary,—that the important characteristic and always-present symptoms of this variety of *coup de soleil* may be summed up as intense fever with profound disturbance both of the cephalic and spinous nervous system; the disturbance manifesting itself in the form of insensibility, with or with-

out delirium, and with restlessness, convulsions, or paralysis of the motor tract.

The following notes of cases were all taken during my residency in Pennsylvania Hospital, and, having been made before I was conversant with the literature of the subject, or had thought much about it, are unfortunately not so full on certain points as is desirable:

CASE I.—T. B., Irishman, æt. about 30, was brought into the ward at 1 P.M. August 2, 1863. He was said to have fallen whilst walking in the street a few minutes before. He was perfectly unconscious, with very labored breathing. His pupils were not markedly dilated or contracted, and yielded sluggishly to light. His skin was cyanosed and very hot and dry. He had vomited and passed his feces unconsciously. His pulse was 110, quick and moderately strong. He could scarcely swallow. Turpentine injections and brandy were exhibited; but the man died at 5 P.M.

Autopsy, two hours after death.—Rigor mortis marked. Venous trunks of the meninges of the brain loaded with dark blood. Brain substance normal. Lungs congested. Heart rigidly contracted. A thermometer in thorax indicated 108° F. Blood very dark-colored, with a slightly acid reaction; not coagulating. Blood-corpuscles under the microscope normal, but very dark-colored.

CASE II.—Irishman, æt. 64, a moderately stout, muscular man, was brought into the ward about 7 P.M. August 10. His skin was very hot, belly tympanitic, pulse 177, not intermittent, but very weak. He had had involuntary discharges from the bowels. The face was very much con-

torted by spasms, repeated pretty regularly 130 times a minute. He was treated with turpentine injections, brandy, aromatic spirits of ammonia, etc. He died quietly at 11½ P.M.

Autopsy, one hour after death.—Venous trunks of the meninges of the brain loaded with blood. Brain substance normal, not congested. No bloody or serous effusion. Heart rigidly contracted. Kidneys normal. Blood very dark and fluid; of a slightly acid reaction.

CASE III.—German. A discharged soldier; very intemperate; a large, fat man, weighing about 200 pounds. He was brought into the wards at 1 P.M. August 10. Those who brought him in stated that he had fallen suddenly whilst loading a dray, about an hour and a half previously. His skin was of a dark-reddish color, the capillaries refilling very slowly when they were emptied by pressure with the finger, it requiring several seconds for them to do so. His pulse was 170 and upwards, very irregular and intermittent, but not excessively weak or thready. He was perfectly unconscious, but lay absolutely still, without even subsultus tendinum. His pupils were contracted. The conjunctiva not sensitive, and very much congested. His skin exemplified calor mordax. A thermometer placed in the axilla indicated 109° F. His breathing was slow, very labored, and irregular. He had involuntary discharge of feces. He gradually grew worse, and before death bloody, dirty foam trickled from the nose and mouth. Death occurred at 2½ P.M.

Treatment.—Frictions with ice; brandy and ammonia, as much as could be forced down him. Turpentine injections.

Autopsy, one hour after death.—Cadaver intensely hot, very fat, no rigor mortis. Meningeal venous trunks engorged. Brain substance normal. Left heart slightly concentrically hypertrophied, very firmly contracted. Kidneys normal. Blood very dark-colored, fluid, coagulating slightly, forming not more than a grumous mass.

CASE IV.—C. H., Englishman, over 60 years of age, was brought to the hospital at 1½ P.M. August 11. He was said to have fallen in the street one or two hours previously. He was very restless, almost convulsive; breathing labored and noisy; pulse 170, and slightly intermittent; skin burning hot; temperature in axilla 109° F. His pupils were contracted, the conjunctiva dry, non-sensitive, and injected. There was some, but not strongly pronounced, stasis in the capillaries of the skin.

Treatment.—Brandy, aromatic spirits of ammonia, turpentine injections, and rubbing with ice. He died in half an hour.

Autopsy, one hour after death.—Cadaver very fat. Meningeal venous trunks engorged. Brain substance very slightly congested, the ventricles distended, with slightly reddish serum; no effusion of blood. Left heart slightly hypertrophied, firmly contracted. Liver very fatty. Kidneys normal. Spleen very much enlarged, and softened. Bladder empty, rigidly contracted. Blood very dark; coagulating, but not firmly.

CASE V.—J. B. An intemperate Irishman, æt. about 33, was brought into the wards of the hospital at 3½ P.M., August 14. His wife stated that on the 10th he had been so exhausted by the heat, so sick at the stomach, and had suffered so much from headache, as to be forced to give up work until the morning of the 14th. When he entered the ward the skin was very moist, but intensely hot, and covered with a rubeoloid eruption. A thermometer placed between the thighs indicated 104° F. The pupils were slightly contracted, the conjunctiva injected and very sensitive. There had been no discharges. The pulse was 140, and rather feeble. He was entirely unconscious, but was continually muttering unintelligibly, and was very restless. He vomited freely.

Treatment.—Cold water poured by the bucketful over the head and breast, and turpentine injections. At 4 o'clock his restlessness was replaced by convulsions, with very marked opisthotonos. These convulsions lasted some five or six minutes each, and were somewhat epileptiform; but as the secretion of saliva was entirely dried up, he did not foam at the mouth, although his jaws worked violently. His breathing was for the most part very hurried, shallow, and irregular, but at times labored and slow. He passed a few drops of urine, and his bowels were moved by an injection. Brandy was put in his mouth, but its exhibition produced immediately fearful convulsions, probably owing to the difficulty of deglutition.

The cold affusions lowered the temperature of the skin, but did not resuscitate him in the least. At 4½ o'clock the douche was repeated, but this time produced violent spasms, with vomiting and great congestion of the face. From this time his symptoms deepened, his body became very dark-blue or purplish, and he died quietly at 5.15 P.M. No post-mortem was allowed.

CASE VI.—An Irishman, middle-aged, robust, and muscular, but not fat, was brought into the wards at 8 P.M., August 14. He was said to have fallen about 3 P.M. He was perfectly unconscious, somewhat restless, with muscular twitchings and subsultus tendinum. His tongue was very dry; his skin dry, harsh, and hot. The temperature in the axilla was 104° F. His pupils were slightly dilated, his conjunctiva injected; pulse 150, weak, not intermittent, but somewhat fluttering; breathing 48 per minute, and very laborious. He could swallow only with difficulty. He was treated simply with brandy, and died quietly about 11.30 P.M.

Autopsy, one hour after death.—No rigor mortis. Temperature in abdomen 108° F. Brain, with its large venous

trunks, engorged, and ventricles containing an abnormal amount of serum. Left heart rigidly contracted. Liver very fatty. Blood very dark and fluid, with a decided acid reaction.

CASE VII.—C. B., German, a large, muscular man, was brought into the hospital at 12.30 P.M., August 15. The skin was very hot and dry, axillary temperature 109° F.; pupils almost normal, conjunctiva injected, mouth moist, deglutition almost impossible. He had a severe convulsion immediately after his entrance, and died in a very few minutes. There was a large ecchymosis in one axilla. He was said to have fallen whilst working in a sugar refinery, and to have been brought at once to the hospital.

Autopsy, two hours after death.—Meningeal veins gorged with blood. Some serous exudation in ventricles. Left heart rigidly contracted. Temperature in abdomen 110¾° F. Blood decidedly acid, very fluid, without a sign of coagulation. Under the microscope the red corpuscles were apparently darker than normal.

CASE VIII.—An Irishman, only a few days in the country. He was said to have fallen during the latter part of the afternoon, whilst wheeling coal. When brought in at 9 P.M. he was semi-unconscious, but could scarcely speak intelligibly; his pulse was 90 per minute, moderately strong; surface dry, but not inordinately hot; he had no pain, but complained of great weakness; he had not had involuntary discharges.

Treatment.—Ten grs. of muriate of ammonia and f℥ ss of brandy were given every half-hour, and an injection of an ounce of turpentine was administered. At 10.30 P.M. his pulse had fallen to 80, and his general condition much improved. His medicine was directed to be given every hour only.

August 12, he was entirely conscious, but very drowsy, and slept a great deal.

August 14, well. He now states that previously to his attack he had been drinking freely of ice-water, but had not been sweating at all, and that he had no premonitory symptoms, no signs of exhaustion, no optical derangement, no headache, etc.

In many of the cases which have come under the notice of the author of this memoir, the evidences of asphyxia were quite marked some time before death; but at the same time there was generally a consentaneous failure of the heart's action, so that the immediate cause of death was not merely failure of respiration, but also of the heart's action.

These cases, I think, represent the ordinary variety of the disease seen in our large cities.

According to Maclean, Dr. Morehead* has divided insolation into three varieties: the cardiac, the cerebro-spinal, and the mixed. "In the cardiac variety, although it is probable that the sufferer is himself conscious of some premonitory symptoms, there is seldom time for their full development, so as to attract the attention of bystanders, before the patient falls, gasps, and, in some severe cases, expires

* Dr. Maclean gives no reference, and I have not been able to find where the classification is made. As the Royal Society's catalogue does not ascribe to Dr. Morehead the authorship of any especial paper on sunstroke, it seems probable the classification was proposed in his work on the Diseases of India. If so, it must be in a later edition, for the first contains no reference to any such division.

before there is time to do much or anything for his recovery, death taking place by syncope."

This variety I have never seen an instance of, and further discussion of it will be postponed to a later part of this paper.

The distinction between cerebro-spinal and mixed cases is certainly not so evident as the separateness of the cardiac variety. If the pathology hereafter developed be, as it seems, true, there can be no mixture between the cerebro-spinal and the cardiac. Leaving out of view the cardiac, it is true that cases of insolation may be divided into those in which death takes place purely through paralysis of respiration, and those in which the heart also suffers a gradual weakening; but as these cases are not practically—*i.e.* therapeutically—distinct, I cannot see any advantage to be gained by such a separation, especially as cases in which the heart does not suffer more or less are so very rare, that I have not only never seen a case, but do not know of an unequivocal account of more than one or two. The nearest approach to such that I can call to mind is the following by Dr. Crawford (Madras Journ., No. 2):

CASE IX.—A. B., aged 24, a soldier, was heard at midnight moving, and his comrades, thinking the noise strange, cried out, and, not receiving a reply, got up, and found him muttering incoherently about a drink. He became quiet, then comatose, and when I [Dr. Crawford] saw him about

a quarter-past one o'clock, I found him moribund; respirations short, quick, and stertorous; pulse full and bounding; face flushed, eyes suffused, pupils contracted to a point, and skin hot. I had scarcely completed my examination, when all the sphincters relaxed; the contents of his stomach, chiefly water, welled from his throat; a frothy mucus tinged with blood ran from his nostrils; his pupils dilated to their utmost extent; a slight tremor crept over his frame, and he was dead.

In contrast with this, as representing a typical "mixed" case, may be cited the following from Dr. Barclay's paper (loc. cit., p. 368):

CASE X.—R. C., æt. 26; not intemperate; was attacked with fever, while on guard, on the 26th of May, and came into the hospital the evening of the same day. On the morning of the 27th he was quite free from fever, but rather weak, and was detained in consequence. He lay on his cot during the forenoon without making any complaint. About noon was observed to be in a state of insensibility, and breathing heavily. He was removed at once to the coolest veranda in the hospital, the cold douche applied over his head, chest, and back, and eight leeches applied to his temple by the apothecary then on duty.

At 1.2 P.M. I saw him. He was then completely insensible, his face paler than usual, his eyes fixed and slightly turned upwards, the pupils somewhat contracted, but much less so than was usual in the cases in the field. His skin felt burning to the touch. His pulse was frequent and rather full. The pulsation in the carotid was very strong, and could be seen at a distance. He had no convulsions, nor could the slightest movements of his limbs or eyelids be observed for hours. On stethoscopic examination, loud subcrepitant râles were heard all over the chest. The first sound of the

heart was natural, the second indistinct. The leeches, which had drawn very little blood, were removed, and there was scarcely any bleeding from the bites. The cold douche was applied assiduously for some time, but without any good result, and had to be discontinued on account of failure of the pulse. An attempt was made to give a stimulant, but nothing could be swallowed. A purgative enema was given at once, and a considerable quantity of thin feculence was brought away with it. Enemata of brandy-and-water, with from fifteen to twenty minims of chloroform, were given repeatedly, but they were never retained for more than a few minutes, and no effect seemed to be produced by them. A blister was applied to the nape of the neck, sinapisms to the chest and feet, and ammonia to the nostrils. His head and the whole surface of the body were kept wet, and his face assiduously fanned. During the afternoon he became gradually worse. His pulse became imperceptible, his conjunctiva pinky, his hands and feet livid. His head continued for a couple of hours firmly bent backwards, and his hands, forearms, and toes flexed. By evening the blister had risen well, and he then improved considerably. The spasm disappeared, the lividity of his hands became less, his pulse returned, and he regained a certain amount of consciousness, and was able to swallow with difficulty. A small quantity of brandy-and-water was then given every half-hour; and, the bronchial tubes being evidently loaded with mucus, he was occasionally turned over on his face, his head projecting over the edge of the cot. This change of position was generally followed by efforts to vomit, by which his breathing was greatly relieved. About midnight he began to sink again. The insensibility became more profound, and the breathing more stertorous and oppressed. A blister was applied to the vertex without any relief. The pulse gradually failed, and he died at 3.20 A.M. on the 28th.

Whether the division into mixed and cerebro-spinal insolation be accepted or not, I think it must be allowed that, in the majority of cases of the disease, death is induced by asphyxia, especially when it comes on quickly but not instantaneously. The general concurrence of authors in this is indicated by the frequent use of the name "heat" or "solar asphyxia." To corroborate my own observation further, I will make one or two quotations.

Dr. Dowler, of New Orleans, who has watched a very large number of cases from immediately after the fall until death, says (New York Medical Gazette, 1842, pp. 214, 215), "The cause of death begins, continues, and ends in the breathing apparatus. . . . After the death of the lungs or the cessation of the respiration, the heart and arteries will, in some instances, continue to act."

Dr. Pirrie says (London Lancet, May, 1859), "The symptoms are distinctly those of that mode of dying in which the disease commences at the lungs." Dr. Crawford (loc. cit.) goes further, believing even the coma to be secondary upon the asphyxia.

The conclusion seems logically inevitable that, even in very many rapidly fatal cases, death occurs from paralysis of the respiratory centres by the excessive heat or other causes.

There is an affection which is rarely, if ever, seen in this country, but which appears to be very com-

mon in India, where it is known as *ardent continued fever*. This disease is really scarcely worthy of a distinct place in the nosological catalogue, but is a variety, or rather degree, of insolation, arising from the same cause, presenting a similar but less violent array of symptoms, and often passing into the fully-formed *coup de soleil*. One of the earliest, and at the same time clearest, accounts of this fever which I have met with is that in Morehead's work on the Diseases of India. After stating that, although mental excitement, intemperance, etc. are often factors of importance in the production of the disease, elevated temperature is the necessary condition, Dr. Morehead gives the following account of the symptoms:

"The attack is generally sudden, often without much chilliness. The face becomes flushed; there is giddiness and much headache, intolerance of light and sound. The heat of skin is great; the pulse frequent, full, and firm. There is pain of limbs and of loins. The respiration is anxious. There is a sense of oppression at the epigastrium, with nausea and frequent vomiting of bilious matters. The bowels are sometimes confined; at others, vitiated discharges take place. The tongue is white, often with florid edges. The urine is scanty and high-colored. If the excitement continues unabated, the headache increases, and is often accompanied with delirium. If symptoms

such as these persist for from forty-eight to sixty hours, then the febrile phenomena may subside, the skin may become cold, and there will be risk of death from exhaustion and sudden collapse. In most cases the cerebral disturbance is greater in degree, and in these death may take place at an earlier period in the way of coma."

Whether Dr. Morehead has confounded two or more fevers somewhat, I do not know; but Sir J. R. Martin certainly says (The Influence of Tropical Climates on European Constitutions, p. 208), "We have not here the tendency to collapse so characteristic of the true Bengal remittent fever."

A reference to the quotation from the paper of Dr. Bonnyman, already given, will show that he has evidently seen cases of fever, excited by heat, some ending, others not ending, in insolation.

Dr. Barclay (loc. cit., pp. 365, 367, 368) states very plainly that during the hot season of 1858 there were very many cases of men whose systems were in a state of feverish excitement from the heat, others which were more serious and were entered upon the hospital roll as cases of *febris continuus communis*, and others which were marked as *insolation*.

The line which he drew between the last two affections he asserts to have been a purely arbitrary one. Those cases in which insensibility or con-

vulsions were present, were called insolation; others, common continued fever.

This evidence might be increased by further quotations, but is certainly sufficient to show that very often insolation is preceded by an acute ephemeral fever, and that this fever is caused by exposure to heat, and may exist either with or without inducing the symptoms ordinarily known as sunstroke: the difference between the affections is therefore simply one of degree, not of kind.

Pathology.—The post-mortem appearances, after sunstroke, are mostly negative: there is scarcely any constant lesion whatever of the solids, nor is congestion of the brain, or serous or hæmic effusion into its ventricles or substance, of frequent occurrence. All authors appear to agree in stating that the right heart and the pulmonary arteries, with their branches, are gorged with dark fluid blood. In my cases the lungs did not present at all the appearance of congestion of their minute capillaries, but when they were cut the blood poured from them abundantly, seemingly from their larger vessels. Not only do the lungs suffer from venous congestion, but the whole body also. The blood appears to leave, as it were, the arterial system, and collect in the venous trunks. The arterial coats are often stained red, apparently from the altered hæmatin of the blood.

In my autopsies I was astonished to find the heart, especially the left ventricle, rigidly contracted. It had been previously stated by some observers that the heart was soft and flaccid, whilst most had not reported at all the condition of the viscus. There was no room for doubt in the observation; in every case the heart was rigid and hard to a degree which none of us had previously seen. The question at once arose, How is this to be reconciled with testimony? The observations of Dr. Pepper have already been shown not to be applicable to the subject. But there remain those of Levick, who failed to find any rigidity. The cause of this failure is, however, sufficiently obvious. The post-mortems were made from thirteen (Levick, Pennsyl. Hosp. Reports, 1868) to thirty hours after death. As the temperature of the body remains above 100° for hours, it is evident that putrefactive changes, often already entered upon before demise, must go on very rapidly, and that probably even three or four hours would afford sufficient time for the relaxation of commencing decomposition to follow the heat rigidity. Moreover, direct evidence of the truth of this is not wanting. It has been experimentally demonstrated (see Boston Journal of Med., vol. x. p. 350) that in animals rigidity of the heart is found directly after death from excessive heat, but that in a very few hours it disappears.

There can be no doubt that the blood suffers in sunstroke very similarly to what it does in low fevers. Its coagulability is always, so far as my experience goes, impaired, but not always destroyed; and it is probable that in the very rapid cases it may not be decidedly affected. Generally, the blood appears after death as a dark, often thin, sometimes grumous fluid, whose reaction is very feebly alkaline, and in some of the cases herein reported was even decidedly acid. Dr. Levick (loc. cit., p. 373) appears to assert that the blood disks, as seen by the microscope, were shriveled and crenated, and showed very slight tendency to adhere in rouleaux. In several of my cases the blood was carefully examined by the microscope, but nothing abnormal was found.

PART II.

NATURE.

Sufficient of authority and reason has been brought forward to make it at least probable that heat is the sole exciting cause of sunstroke. This being so, it is to be expected that the lower animals, as well as man, should suffer from the affection, and experience fully corroborates this *a priori* reasoning. We are able, therefore, to induce sunstroke in animals, and, by varying its conditions, study its nature much more thoroughly than can be done at the human bedside. A discussion of the nature of *coup de soleil* must, I conceive, rest largely upon such basis of experimentation.

Of the various experimenters upon the effect of heat upon animals, I have had access to, and have used especially, the works of Dr. Vallin, Dr. Stiles, and Claude Bernard. The observations of these gentlemen, in so far as they cover the same ground, are in close agreement; and I myself have attained similar experimental results, although I have gone

further and have read the phenomena somewhat differently.

The symptoms produced by exposing an animal to excessive heat do not appear to differ save in degree, whether the heat be artificial or due to the direct rays of the sun, or whether it be moist or dry. The animal is at first excited, trying to get away from the cage in which it is confined. This period of excitement sooner or later, according to the intensity of the heat, gives place generally to a second stage of profound muscular prostration and quietude. From the beginning the respiration has been exceedingly hurried, and now very often it cannot be counted. The beat of the heart keeps pace with the respiration, and panting and exhausted the animal lies quiet, with the saliva pouring from its open mouth. This second stage soon yields to that of coma. In my own experiments the insensibility has generally come on gradually, not suddenly, and has not been accompanied by convulsions. In one case, however, the animal was attacked by coma and fatal convulsions with absolute abruptness.

According to Dr. Vallin's experience, convulsions are very commonly present.

Claude Bernard recognizes them as sometimes present, sometimes absent; whilst Dr. Stiles says that occasionally life is terminated by a fit of convulsions, but generally it ceases gradually.

The resemblance between these symptoms and those of *coup de soleil* of man is very marked. There is in many cases of the latter the same stage of feverish excitement, the same condition of weakness, the same gradually approaching coma. In other cases in man the nervous symptoms come on suddenly, as they do sometimes in animals. The chief difference appears to be, that whilst in animals sudden onset of the nervous symptoms is rare, in man it is common, the reason evidently being that the cephalic nervous system of man is much more delicate and more easily disturbed than that of animals. The post-mortem appearances are very similar to those seen in man. The arterial system is comparatively empty, and there is absolute engorgement of the large venous trunks with blood, whose crasis is more or less broken down.

The muscular system offers the most obvious lesion, and it is to this I shall first direct attention. Dr. Stiles says (1864), "When the thorax is opened (just after death) the ventricles of the heart are found rigidly contracted, or become so in five or ten minutes, both auricles distended with blood, and manifesting fibrillar movements. The cut surface of the ventricles is acid to litmus-paper; under the microscope the muscular fibres of the heart have lost their transverse striæ."

Dr. Vallin says (1870), "At the moment of death

the ventricles are contracted upon themselves, globular; the left ventricle in especial has a ligneous hardness. Its cavity is effaced, and does not contain a trace of liquid blood, nor of clot. The auricles—the right especially, and sometimes the right ventricle—are full of black liquid blood, or confluent clots. The ventricles instantly after death are insensible to every kind of stimulus. The auricles have for a very few minutes spontaneous rhythmic movements, but in a little while these cease, and cannot be reprovoked; all the muscular tissue is strongly acid."

Claude Bernard says (1871), "In opening the cadaver immediately after death there is found generally an arrest of the beating of the heart, a black coloration of the blood in the arteries, sometimes ecchymosis under the skin, and lastly cadaveric rigidity comes on with great rapidity" (loc. cit., p. 139). Claude Bernard and Vallin, then, agree in stating that the voluntary muscles are affected similarly to the heart, and that the muscles directly after death either do not react at all to the stimulus of galvanism, or do so very feebly and imperfectly, and that post-mortem rigidity comes on almost immediately after death. Vallin says, "The diaphragm is, after the heart, the muscle which loses its irritability most quickly: generally at the moment of death it fails to contract under any stimulus. Certain muscles of the trunk commence

to become rigid even during the agony, and in about half an hour this rigidity is generally complete for the whole body. A constant accompaniment of the muscular rigidity is an acid reaction." There can be no doubt that in some instances this rigidity is almost consentaneous with death. Dr. Stiles, however, states that in his experiments on rabbits post-mortem rigidity did not come on until between one and two hours *after* death.

As a study of the subject, the following experiments were performed. The first two of them were made in the last week of September, when the sun had already waned in power, without the aid of artificial heat. A box was constructed rudely with a slanting glass lid, like a miniature green-house. It was simply placed upon a brick pavement, when used, in such a way that the sun could exert its fullest power upon it.

EXPERIMENT 1.—Exposed a two-thirds-grown rabbit in a box covered with glass.

1 P.M.—Temperature in rectum, 104½°. Temperature of box, 120° (F.).

1.15 P.M.—Temperature, 106½°. Respiration very hurried.

1.30 P.M.—Temperature, 109½°. Has convulsive attacks, in which he jumps, and kicks with hind legs with great fury.

1.45 P.M.—Temperature, 112°. Seems very weak and relaxed; breathing 220 a minute. Lies on side, with every now and then the attacks alluded to; slobbering greatly.

2.10 P.M.—Temperature of box, 120°. Rabbit on side, exceedingly weak, gasping; squealing faintly at intervals.

2.15 P.M.—Temperature, 114½°. Perfectly unconscious; lies relaxed and motionless on the cool ground in the shade.

2.20 P.M.—Only gasping at long intervals; heart still beating, although laboredly, and somewhat irregularly, yet pretty steadily, and with some force.

2.21 P.M.—Dead.

Autopsy.—Heart: right side and left auricle full of blood; left side, containing blood, not contracted. The heart made a few very imperfect and feeble attempts at beating when it was cut across. Blood coagulating with great rapidity and firmness; alkaline. Brain not congested. Muscles all failing to show the slightest sign of contraction under the strongest faradaic current, except some of the leg muscles, which contracted very feebly, and only at all when the current was very intense.

EXPERIMENT 2.—Two-thirds-grown rabbit. Put in a box at 11.30 A.M. Temperature, 104½°.

12 M.—Temperature, 109°. Temperature of box, 112°.

12.25 P.M.—Temperature, 110½°. Rabbit weak, slobbering a great deal; breathing with great rapidity.

1.15 P.M.—Rabbit conscious, lying quietly on his side; not slobbering; breathing not nearly so rapid, but deep and labored. Temperature, 111½°.

1.35 P.M.—Rabbit found dead. Temperature, 112°.

Autopsy.—Heart: left ventricle empty, very firmly contracted, with a very evident white spot at apex. Galvanic (induced) current very strong, giving rise to no muscular movements whatever, either of heart or voluntary muscles. Blood coagulating slowly and imperfectly; reaction neutral, or at least so feebly alkaline as to be uncertainly so. Muscular reaction very decidedly acid. Spinal cord not congested. Right side of heart gorged with blood.

EXPERIMENT 3.—A large adult rabbit.

12.11 P.M.—Anal temperature, 105°. Just put in box, whose temperature is 130°, heated by very hot brick flues, on which the rabbit lies.

12.15 P.M.—Rectal temperature, 107°. Breathing excessively hurried.

12.17 P.M.—Rectal temperature, 109°.

12.21 P.M.—Rectal temperature, 111°. Had a moment since what was apparently a convulsion, and has had numerous convulsive twitchings since. Appears semi-unconscious.

12.25 P.M.—Dead. Temperature in abdomen after death, 111°. Respiration ceased some time before heart. The thorax was opened, and the heart was felt with the finger to be pulsating. It was then further opened, and the heart was seen to be very distinctly pulsating, and gradually becoming filled with dark blood. It was punctured, and blood allowed to escape; it made one or two pulsations, and then at once became rigid. After this the diaphragm was tried with the galvanic current, and responded to it. The muscles of the hinder extremity failed to do so, those of the fore legs did. Peristaltic action of the intestines was moderately active when the body was opened, and on galvanic excitation became very active.

EXPERIMENT 4.—A moderate-sized dog.

1 P.M.—Fut in the hot box.

1.15 P.M.—Anal temperature, 106°.

1.30 P.M.—Anal temperature, 110°.

1.40 P.M.—Anal temperature, 110¾°. Just dead.

Autopsy.—As soon as respiration ceased, the body was opened. Heart still beating, gorged with dark blood. Veins full of dark blood. Blood on being shaken in test-tubes rapidly clotting, and slowly changing its color to an

arterial hue. The vessels were carefully examined: no clots were found in them.

EXPERIMENT 5.—An adult pigeon.

11.40 A.M.—Rectal temperature, 109°. Just put in box; the temperature in box, 130°; besides, the pigeon was in direct contact with the very hot brick flue.

11.45 A.M.—Respirations very weak.

12 M.—Has been unable to stand for some time; has been semi-unconscious. Just had a convulsion, followed by persistent opisthotonos.

12.2 P.M.—Anal temperature, 120°?. (My thermometer did not mark higher than 120°, to which the mercury rose; the hand could hardly bear the heat of the flesh.) Dead. Respiration certainly ceased before the heart's action. Rigidity came on almost before heart ceased beating. Thorax opened as soon as heart ceased action. The heart was found rigidly contracted like a board. Muscles acid.

EXPERIMENT 6.—Precisely like last in conditions and results. Death temperature the same.

EXPERIMENT 7.—Precisely like last in all respects. Death temperature the same.

In none of these experiments was the thorax opened before the heart ceased to beat.

EXPERIMENT 8.—A young pigeon, put in box at 130°, and taken out some time afterwards dying.

The heart being felt to be beating after respirations had ceased, the body was opened and the pulsations were watched for some time.

EXPERIMENT 9.

12.36 P.M.—Anal temperature, 110¾°. Pigeon put in hot box.

1 P.M.—Anal temperature, 113¾°.

1.30 P.M.—Pigeon found dead; on its being opened, the heart was found to be beating, although irregularly and very feebly.

EXPERIMENT 10.—Cat.

12.16 P.M.—Temperature of box, 130°.

12.35 P.M.—Temperature of box, 130°. The cat ever since having been put in the box has been struggling violently and savagely, and for the last five minutes has been evidently growing weaker, but perfectly conscious. Just seized with a sudden tetanic convulsion, which instantly arrested all respiration, and persisted with absolute rigidity for about five minutes (not by actual timing). When the cat was taken from the box the pupils were widely dilated, the heart beating strongly and regularly. She was plunged into cold water, but never made an effort at breathing, although perfect relaxation of the muscles soon came on. The body was opened; heart found to be still beating and distended with blood; after a considerable time it was seen to gradually stop beating, and the left side contracting expelled all the blood from it, and became rigid under our eyes. The diaphragm responded, though somewhat feebly, to the galvanic current fully fifteen minutes after respiration had ceased.

These experiments confirm in many respects the results of previous observers, but they also show that the *rigidity of the heart and of the muscles is often not ante mortem, but post mortem*, occurring sometimes at the moment of death, at other times not

until some minutes after it. In all those cases in which an especial observation of the point was made, respiration ceased before the heart's action. In Experiment 8, the pigeon was opened directly after death, and the pulsations of the heart watched for some time. In Experiment 10 the same thing was done upon a cat. In this case, the heart underwent the process of hardening beneath my view. The change was a quick but not instantaneous one; the viscus appeared gradually to shorten itself, and to become at the same time less broad, and put on a very rigid look,—the whole process taking perhaps about half a minute to complete itself. This experiment was also interesting as showing that the diaphragm does not lose its functional power before death, nor is always the first muscle in the body to do so.

Experiment 10 was of great interest on account of the sudden seizure resembling so closely certain forms of sunstroke in man. The symptoms were evidently not of muscular origin, the heart persisting in its regular beat long after respiration had been arrested by a tetanic spasm, from which the muscles after a time completely relaxed, and the diaphragm responding to electricity fully fifteen minutes after the cessation of respiration.

Muscular System.—As the very rapid post-mortem rigidity of the voluntary and involuntary muscles is

certainly among the most prominent and characteristic lesions of sunstroke, it is evident that a study of the causes or nature of post-mortem rigidity is most vital to a discussion of the nature of the affection.

Three theories have been advanced to explain this rigidity. The first is, that it is a sort of vital contraction, allied to the normal contraction, which has become permanent. This vitalistic idea has been recently placed in its most definite, tangible form at the hands of Chas. Bland Radcliffe, who has formulated the following proposition: "Rigor mortis, like ordinary muscular contraction, would seem to be caused by the elasticity of the muscular fibres being allowed to come into play by the disappearance of the electrical charge which had previously kept these fibres upon the stretch, the contraction in rigor mortis being permanent simply because this disappearance is final." Another vitalistic theory, essentially different from that of Radcliffe, is the so-called idio-muscular theory of Schiff, according to which an acrid acid juice is developed by a commencing decay of certain portions of the muscles, and acts upon the remaining sound portion as a stimulant, causing persistent contraction.

It would be digressing too widely to discuss the general theory of Radcliffe as to muscular contractility. It suffices for my purpose to deduce the following objections, which, in the absence of

any proof in favor of it, seem to me fatal to a theory which associates normal vital contractility and post-mortem rigidity.

The *physical conditions* of the contracted muscle are very different from those of the rigid one. Thus, the elasticity of the two is entirely different: the contracted normal muscle stretches, the rigid one breaks. Kühne has shown, and Harless (Untersuchungen an der Muskelsubstanz; Sitzungsberichte der bayer. königl. Akad., 1860) has confirmed, that whilst the tetanized muscle is translucent, the rigid one is turbid and opaque, and the turbidity has been proven to come on *pari passu* with the rigidity. Dr. Harless experimented so as to show beyond cavil the simultaneous occurrence of opacity and rigidity, in the following way. A piece of frog's muscle was tightly placed between the compressorium plates under a microscope, and a stream of light thrown through it just sufficient in power to bring out distinctly the lines of a cobweb micrometer in the eye-piece. It was found, on heating the fragment of tissue, that at from 36° to 40° Cels. (=96⅘° to 104° F.) the cobwebs were lost to sight from the muscle becoming less transparent (loc. cit., p. 128).

Herrmann and Walker, who have recently (Pflüger's Archiv, ii. 182, 195) called attention to the difference in the contraction of post-mortem rigidity and that of living muscle, state that whilst the living

muscle will raise a weight much higher than occurs during post-mortem hardening, during the latter process a heavier weight will be lifted than the most powerful electrical stimulation will induce the living muscle to elevate.

The second objection to the theory of identity of rigor mortis and muscular contractility was first urged, I believe, by Dr. Harless (Untersuchungen über die Muskelstarre, Sitzungsberichte der königl. bayer. Akad., 1860, p. 456.) It is as follows: If the loss of irritability of a separated muscle be expressed by a curved line, the curve at first sinks gradually and towards the close abruptly, or, in other words, the irritability at first lessens very slowly, afterwards very rapidly. Now, if, as the vitalistic theory contemplates, the irritability and post-mortem rigidity are, as it were, complementary, a curved line expressing the growth of the latter ought to be similar to that expressing the growth of the former,—*i.e.* a curve at first gradual, afterwards sudden. This, however, does not happen, but from the beginning the muscular currents, which are an inverse index of the post-mortem rigidity, lessen with decreasing rapidity. It would appear at first as though Morgan (Electro-Physiology and Therapeutics, p. 327) expressed a different belief from this of Harless as to the relations spoken of, for he says, "The diminution of the muscular current is directly as the de-

crease of the irritability of the muscle." It appears, however, that Dr. Morgan does not mean this to be construed too literally, for in his diagram (Fig. 101) the curves representing the loss of muscular currents follow the law of Harless.

Kühne also found by experiment that the expressed acrid juice of dead frog muscle has no power to cause contraction of living frog muscle. Evidently a *coup de grâce* to Schiff's theory.

As early as 1842, Brücke (Ueber die Ursache des Todtenstarre, Müller's Archiv, 1842) accounted for the occurrence of post-mortem rigidity by the coagulation of a plasma in the muscle; he, however, failed to prove his supposition. The difficulty he met with was, that whenever he attempted to press out the plasma from the muscle, the change would take place whilst the latter was in the press, and he would obtain only rigid muscles and an acid juice.

In 1859 Kühne took the matter in hand (Untersuchungen über Bewegungen und Veränderungen der contractilen Substanzen, Reichert und Du Bois Reymond's Archives, 1859, p. 564), and succeeded in overcoming the difficulty by the use of salt and cold, both of which agents have the power of retarding muscular rigidity. He first washed the blood out of the muscles of frogs by injecting through the arterial system of the animal just killed a one to two per cent. solution of salt; then cut the flesh into small pieces, washed it with salt water,

and put it in a cold press. By this means he obtained a glairy fluid, which near the freezing temperature remained liquid, but at 40° C. (104° F.) instantly formed a large, firm, translucent coagulum. At a temperature of 35° C. (95° F.), coagulation occurred in an hour; at 38° C. ($100\frac{2}{5}$° F.), in half an hour; and at from 12°–14° C. ($53\frac{3}{5}$–58° F.), at the end of six hours.

Kühne also performed similar experiments upon a rabbit with similar results, obtaining a glairy liquid, which coagulated after standing three or four hours at an ordinary temperature.

I have repeated the experiment of Kühne on a rabbit, with results similar to his.

EXPERIMENT 11.—November 15, 1871. A full-grown rabbit was killed by a blow on the back of the neck. It was opened immediately, and salt water was thrown into the descending aorta so as to come out of the vena cava ascendens. The muscles were then cut into small pieces, thrown into salt water, and put immediately into a press. The liquid first obtained was rejected; it was thin and pinkish; afterwards, under strong pressure, a clear, slightly pinkish viscid fluid was procured. The reaction of this was neutral; but on exposure of the wet litmus-paper to the air, the acid reaction was soon developed. This fluid was gradually warmed, and when 115° F. was reached at once changed color, became whitish and somewhat turbid; at 116° F. there was decided coagulation. If 110° F. was maintained, in the course of a very few minutes coagulation took place. After coagulation the reaction was decidedly acid.

Dr. Pickford (Henle und Pfeuffer's Zeitschrift für ration. Medicin, Neue Folge, i.) was, I believe, the first to notice that a frog's muscle would become rigid when placed for a few seconds in a liquid heated to 65° R. (= 178¹° F.), or for some minutes in one at 30° R. (= 99¼° F.).

Schiff (Lehrbuch der Physiologie) and Mundt (Die Lehre der Muskelbewegung) confirmed this, but to Kühne belongs the credit of having first thoroughly investigated the subject, in the paper already quoted. He found (loc. cit., p. 789) that a muscle of a frog plunged into a liquid of 65° R. or 82° C. (= 178¼° F.) for twenty-five seconds became permanently rigid; and that if a muscle were left for a whole minute in a liquid at the temperature of about 45° C. (= 113° F.) the same result followed. Further, that if the muscle were immersed for some minutes in quicksilver at a temperature of 37.5° C. (= 100° F.) it lost its power of contracting under the influence of the strongest galvanic stimulus, and yet remained limp, and with an alkaline reaction. By a still longer immersion it became perfectly rigid. M. Kühne also states that immersion of a frog's muscle in quicksilver at a temperature of 40° C. (= 104° F.) produced almost instantaneous rigidity. This was also found to take place in a living frog when the legs were placed in quicksilver at 40° C. (= 104° F.); and although the circulation was perfectly re-established,

and the frogs kept alive, the muscles never in any sense recovered themselves,—never regained any contractile power. The lowest temperature by prolonged exposure to which the frog's muscles lose their functional ability is between 35° and 37° C. (= 95° and 99° F.).

On the other hand, an exposure of the frog's muscles to a low temperature protracted the period of sensitiveness to stimuli, so that under such conditions the muscles would remain active for days.

With these facts the coagulability of the muscle fluid was found in complete accord. Near 32° F. it remained clear and liquid for days; at 40° C. (= 104° F.) it instantly coagulated; at 30° C. (= 86° F.) coagulation occurred in an hour; at 38° C. (= 100⅖° F.) in half an hour. The reaction of the heated muscle was acid, and it was also found that a frog's muscle which had undergone spontaneous rigidity, or one that had become rigid by the sudden application of a heat of 40° C. (= 104° F.), could be rendered more rigid by a heat of 45° C. (= 113° F.). Kühne also found that, even if the frog's muscle had commenced to decay, it was changed (by a heat of 45° C.) into a hard, whitish mass, and also that he could press out from thoroughly rigid muscles an acid juice, which had no tendency to coagulate at ordinary temperatures, but which does undergo immediate coagulation at 45° C. These facts certainly seem to

show that whilst the rigidity produced by a temperature of 40° C. (= 104° F.) in a frog's muscle is the same in nature with post-mortem rigidity, that produced by a temperature of 45° C. (= 113° F.) is of a different nature, and depends upon the coagulation of a distinct albuminoid substance.

In the Sitzungsberichte der königl. bayer. Akademie der Wissenschaften, June, 1860, Herr E. Harless published the elaborate paper which I have already quoted (see page 48), in which he corroborates in the main the statements of Kühne. He succeeded without trouble in obtaining the muscle plasma *musculin* in quantity, and found it a nitrogenous substance more closely allied to casein in its behavior with reagents (p. 101) than to the other albuminoids. He also found that the coagulation temperature is in warm-blooded animals near 45° C. (= 113° F.), and that it varies in different animals, and in the same species under different circumstances (p. 100). Thus, in different rabbits he found it ranged from 42°–48° C. (= $107\frac{3}{5}$°–$116\frac{3}{5}$° F.); in frogs, from 30°–44° C. (= 86°–$111\frac{1}{5}$° F.); in a calf, from 45°–46° C. (= 113°–116° F.); and whilst in a very old cat 45° C., in a newly-born kitten it was 70° C.* (= 158° F.)!

He also found (p. 111) that the formation of the

* This is evidently a misprint.

acid precedes the commencement of coagulation, taking place 4°–5° Cels. (8°–10° F.) below the temperature that the latter occurs at, and occurring sooner when the fluid is allowed to coagulate spontaneously. When the acid forms it alters very materially the color of the liquid, and by setting the latter aside in a glass vessel in a cool room he was enabled to watch the change into a deep reddish-brown liquid, occurring in from twelve to twenty-four hours, whilst not a trace of turbidity existed. This change of color I myself have noted as occurring just before coagulation, and think therefore that Morgan (loc. cit., p. 329) is mistaken when he says the coagulation of the muscle fibrin precedes this acidity; a mistake which might very well have been a mere slip in the manuscript, and it is well known he did not live to read the proof. In a second paper in the Sitzungsber. der k. bay. Akad. d. Wissensch., 1860, entitled "Untersuchungen über die Muskelstarre," Harless attempts to establish the theory that although coagulation of the myosin and post-mortem rigidity occur at the same temperature and consentaneously, the latter is not due to the former, but to a physical action of the acid developed upon the muscular fibrillæ, shrinking them up. I do not think, however, he succeeds in establishing this, and believe that it is disproven by the facts that in some muscles the acidity does not develop (see Morgan, loc. cit., p.

329), and that washing the acid out of a muscle by an alkaline solution will not restore its contractility when rigidity has fully set in. There can be no doubt, however, of the close connection between the acidity and the coagulation of the myosin. A circumstance illustrating this, mentioned by Morgan (loc. cit., p. 333), is the action of a ten per cent. solution of common salt on a rigid muscle. This dissolves out the myosin, and leaves the muscle limp and translucent, and at the same time free from acid reaction.

The only objection to the coagulation theory worthy of notice is found in the circumstance that muscle which has become rigid after the cutting off of the circulation from it, can be made to regain its pliability and contractility by removal of the arterial ligature, or that muscle which has become rigid after the death of the animal can be restored by the injection of warm blood. As early as 1834, Dr. Jno. P. Kay (Treatise on Asphyxia, p. 143) noted that if the circulation was arrested in a limb, the muscles soon lost their power of contracting under the influence of galvanism, but recovered it when the obstruction to the passage of the blood was removed.

His experiments have been repeated and amplified by various observers, especially by Brown-Séquard (Comptes Rendus, June, 1851) and Prof. Stannius, of Rostock (Archiv für phys. Heilkunde,

1852). The experiments of the first of these observers were made both on man and animals; the details it is needless here to speak of, but the results arrived at were, that cadaveric rigidity in animals could be overcome after it had lasted from ten to twenty minutes, and in man a muscle which had been rigid for nearly two hours could be restored. I have not seen the original account of Prof. Stannius's experiments, but, as quoted, they are very similar to those of Brown-Séquard, and the results arrived at only differ in so far that Stannius succeeded in restoring the hind legs of a puppy which had been stiff and cold for one hour. It is this possibility of restoration which has been urged as an objection to the plasmic theory. As stated by Kühne, however, it does not seem to be a fatal objection. The first coagulation of the muscle plasma does not necessarily imply the immediate death of the fibre. And it is conceivable that while in a condition of partial coagulation, as it were, the plasma might yield to the solvent powers of the warm alkaline blood and the renewed activity of the surrounding life forces, and be brought into such a state as to be removed, especially as it has been shown that the myosin can be washed out and the muscle rendered limp by a strong solution of salt. In Brown-Séquard's experiments it was found that the blood changed its color from arterial to venous during its passage through the arm,

showing that vital action was recommenced. Further, it is implied by the experiments both of Brown-Séquard and Stannius, and definitely proven by those of Kühne (loc. cit.), that there does come a time when the rigid muscle cannot be restored, although through the unbound artery the blood courses into the capillaries and veins with steady, free, persistent current. If the contraction were a vital phenomenon, this could not be, because the muscle should relax as soon as dead, and, not being dead until relaxed, whilst still rigid ought to be capable of restoration. In this view, then, the assumed objection is rather a confirmation than a valid objection to the plasmic theory.

This being so, I feel forced to accord assent to the proposition, *that thermic and post-mortem rigidity are alike due to the coagulation of a plasma in the muscles.*

This proposition being acceded, the question naturally presents itself, Is death in sunstroke due to the coagulation of the plasma by heat?

It is evident that in determining this question at least two lines of approach are open,—the one, that of absolute ocular and palpable examination, determining whether or not the heart does stop before respiration, and whether the muscles are absolutely dead before general death; the other, the comparative study of the temperatures at which

coagulation of myosin occurs, and of the death temperature in fatal heat or sunstroke.

Along the last of these roads I shall first approach. Vallin (loc. cit., p. 143) believes that coagulation of the myosin is the cause of death. He says,—after detailing the fact that muscles always become rigid at 45° C. (113° F.),—"The death has always taken place at the precise moment that the temperature of the blood attains 45° C., and a thermometer introduced into the cavity of the heart did not vary more than some tenths of a degree above or below this point; so that it affords, as it were, a sort of natural realization of the coagulation of a muscle by plunging it into water at 45° C. The diaphragm, after the heart, is the muscle which becomes rigid the most quickly, and all the world acknowledges, after Claude Bernard, that the point of greatest heat in the body is where the vena cava traverses the diaphragm. It is easy, then, to understand why at death the heart and diaphragm are rigid, whilst the muscles of the trunk preserve their contractility. Coagulation commences with the left ventricle, which becomes rigid and immovable; its cavity is effaced, and hence is explained the relative arterial anæmia which all the organs present. The right ventricle becomes rigid more slowly and irregularly, and preserves in some cases for a time its activity, so that the blood of the pulmonary circulation, not finding access to

the left ventricle, accumulates in the lungs, producing an enormous congestion, and sometimes rupture. Further, the inertia, or, to speak more accurately, the rigidity, of the diaphragm interferes with the respiration, and adds the effects of asphyxia to those produced by stoppage of the heart."

The temperature at which myosin coagulates has already been shown to vary, but to be in warm-blooded animals often above 45° C. or 113° F. The testimony of Vallin, that the death temperature agrees with this, has just been cited, and in another place in his memoir (p. 139) he says the temperature at the moment of death varies from 44° C. to 46.1° C. Claude Bernard states that the death temperature ranges from 43° C. (109⅖° F.) to 45° C. (113° F.). Dr. Stiles says the temperature never rose above 115° F., and was often one or two degrees below it.

The testimony of Vallin must be received with some guardedness, as he is so fully committed to the support of a theory. It will be seen that the experiments of both the other authorities indicate that whilst in some cases 113° F. is attained, in many others the death temperature is much below it. My own experiments, already detailed, confirm this testimony, the rectal temperature at death varying from 111° to 114° F.

Dr. T. Lauder Brunton, in experiments reported in St. Bartholomew's Hospital Reports, vol. vii.,

states that the upper limit at which the heart, when heated, stands still varies in different cases, but that in the stronger animals it is between 113° and 114° F., and even above. He has seen the heart, however, beating actively at 115° F., and still under the influence of the vagus nerve. These temperatures, however, he remarks, were taken in the rectum, whilst the arrangement of the heating apparatus was such that the probabilities are the temperature was somewhat less in the thorax than in the abdomen. His results, I think, are further vitiated by the fact that the animals were all narcotized either with chloral or opium. In pigeons the fatal temperature was always up to 120° in my experiments; but there can be no doubt that the coagulation point of myosin in these birds is higher than in mammals, as their normal temperature is so much higher. They must therefore be left out of consideration in discussing the question in regard to mammals.

From these different views of the subject it may be safely inferred that in some cases where an animal is killed by overheating, the temperature reached during life is that at which myosin removed from the body coagulates, but in others it is a degree or two below the point.

Further, it seems very probable that the myosin influenced by life forces, and by the steady alkaline current of the blood, would resist a heat in living

muscle which would coagulate it instantly under other conditions. Confirmatory is the statement of Dr. Brunton, already cited, that he has seen the heart beating fairly and still under the influence of the vagus when the thermometer in the rectum indicated 115° F.

Looking at the question under consideration solely from this point of view, the answer is somewhat doubtful, but appears to be that in some cases it is very possible that death of the animal may be due to direct coagulation of the cardiac myosin by heat, whilst in other cases it is most improbable.

It is very possible that the mode of applying the external heat, and especially the intensity of the latter, may affect the result. Certainly my own experience is much opposed to the idea that a bodily temperature of 115° F. is often attained in killing cats and rabbits by heat.

The second means of deciding the question leads in a more positive manner to a definite conclusion. My own experiments were carefully made with a view of determining the matter, and were very uniform in the result.

In no case that was closely studied did the heart cease to beat before arrest of respiration. In Experiment 1 the heart beat very feebly and imperfectly after the opening of the cadaver; in Experiment 2 the animal may have been dead fifteen minutes before the autopsy. In Experiment 3 the

heart was found beating quite actively after death, and was seen to become suddenly rigid. In Experiments 4, 5, and 6, on pigeons, the pulsations of the heart could be felt continuing after the cessation of respiration. In Experiment 7 the thorax was opened and the regular beat of the heart ocularly demonstrated after respiration had ceased. In Experiment 8 death took place from a centric convulsion, and the heart went on for some ten minutes with a strong, vigorous beat.

Further, if it be true that an animal dies from coagulation of the myosin of the heart, it is very apparent that if, by any artificial means, it can be cooled before such coagulation has occurred, whilst the heart is still beating, recovery must result, or at least death be averted for the time.

That an animal may, however, be cooled down to its normal temperature, whilst the heart is beating, and yet the fatal result be in no very perceptible degree postponed, is shown by the following experiment:

EXPERIMENT 12.—Pigeon put in hot box at 1.53 P.M. Temperature, 114°. At 2.14 the temperature was 117°. The pigeon was now taken out of the box very ill, unable to stand, but with the heart beating regularly. It was plunged into cold water, and the temperature reduced to 105°. It did not, however, revive any, and died in some ten minutes.

The results of my experiments viewed alone are, then, very positive, that death does not generally

occur from coagulation of the cardiac myosin, but from arrest of respiration.

Claude Bernard (loc. cit., p. 134), on the other hand, says, " It appears still very possible, at least probable, that there is a coagulation of the myosin, and that this is the cause of the death of the muscles, and of the heart in particular. The manner in which heat causes death is, then, perfectly explained. However, as the animals can be taken from the stove a few moments before the fatal issue and not die for many hours, or even for many days, it seems necessary to admit that there are many other grave alterations produced by the heat."

Modifying my own results by those of Claude Bernard and Vallin, I think the following proposition expresses the truth:

Death in the lower animals from sunstroke or heat fever, not produced by a sudden intense heat, occasionally is the result of coagulation of the cardiac myosin; but in many instances, and probably in the majority of cases, is not so produced, but is the result of arrest of respiration.

If it be true that when an animal is kept in the intensely heated atmosphere until death, it succumbs often from other causes than the coagulation of the muscular plasma of the heart, it would seem that much more frequently would man suffer and die from these other causes. His nervous system,

especially the cephalic, is so much more susceptible that it would be expected to suffer more than that of the animal. More than this, in the experiments I have detailed, the external heat is steadily kept up until death, and hence the body of the animal attains the maximum temperature. Not so with man; very generally the external heat is removed, but death takes place nevertheless.

It is necessary, therefore, to study the effects of heat on man separately from that on animals, to determine whether death does result from coagulation of the myosin of the heart; and I shall approach the subject as before.

First, then, what temperature is attained in cases of insolation or *coup de soleil?*

Unfortunately, very few close clinical records have been kept which can throw light on this point. In India, especially, the suddenness and epidemic nature of the affection, the fact that it mostly occurs during the most laborious and trying periods of campaigns and marches, and the circumstance that the doctor is himself generally on the border of an attack, all combine to prevent any clinical work beyond what is necessary for the safety of the patients.

The following table, taken from Dr. Levick's paper in the Pennsylvania Hospital Reports for 1868, represents, however, sufficiently the disease as it occurs in the United States:

TABLE OF TEMPERATURES.

Case.	*Place.*	*Observer.*	*Temp. Max. observed.**
1.	New Orleans.	Dowler.	113° F.
2.	"	"	109°
3.	"	"	104° after death.
4.	"	"	110° after death.
5.	"	"	106°
6.	Philadelphia.	Levick.	109.5°
7.	"	"	109°
8.	"	"	106°
9.	"	"	105.5°
10.	"	"	112° after death.
11.	"	H. C. Wood, Jr.	108°
12.	"	"	108° after death.
13.	"	"	109°
14.	"	"	104°
15.	"	"	106°
16.	"	"	109°–110° after death.

An examination of this table will show that in no case was a temperature anywhere near 115° F. reached, either before or after death; and that in the large majority of cases the highest point attained was not within six degrees of that degree; although it is very probable that the temperature given does not always represent the actual maximum, yet the evidence seems irresistible that in the majority of cases of insolation in man the bodily heat does not become sufficient to coagulate the cardiac myosin. The results of observation are in close concord with this conclusion. Earlier in this

* Proof seems wanting that the temperatures noted were actually the highest attained in all cases.

paper I discussed the modes of death in sunstroke, and it was, I think, clearly proven that in the generality of cases death is immediately produced by a failure of respiration, or by a gradual consentaneous failure of respiration and circulation.

The conclusion seems to follow inevitably that in the ordinary forms of human sunstroke death is not due to the coagulation of the cardiac myosin, and that the latter change and consequent hardening of the heart is a post-mortem phenomenon, occurring directly after death.

In the discussion of the clinical history of sunstroke, it was stated that there is a distinct class of cases in which syncopal death occurs suddenly. A study of these cases is evidently here in order.

It is a fact well known to sportsmen that rigor mortis comes on with extraordinary rapidity after severe muscular exertion. A rabbit or hare which has been run for several miles by a dog will, on being shot, perhaps, roll over once or twice and be picked up perfectly stiff. The same thing has been observed in the tetanus produced by strychnia, the spasmodic muscles being said to sometimes become rigid even without relaxation. Of these facts there can be no doubt, as they are testified to by every investigator. (See Hermann, Grundriss der Physiologie des Menschen, Berlin, 1870, p. 228.)

The explanation of this, if the theory of post-mortem rigidity herein advocated be correct, is

that the myosin under such circumstances undergoes some peculiar change by which its tendency to coagulation is increased.

Dr. Harless (loc. cit.) has experimentally proven this to be the case, for he found that myosin prepared from a rabbit tetanized with strychnia coagulated at 42° C. (107⅗° F.), whilst that from a similar non-tetanized rabbit did not change until 48° C. (118⅖° F.) was reached.

Certain experiments which I have performed would seem to illustrate this fact by its opposite. In Morgan's work (loc. cit., p. 332), the following method of preparing myosin in large quantities is given: "Enough common salt in powder is mixed with snow in a cold vessel to form, when thawed, a one per cent. solution, and this mixture rubbed up intimately with one-fourth of its weight of shavings of frozen frog's muscle is, while still at 3° C., thrown on a piece of linen; then the liquid is filtered several times through paper wet with a cold one per cent. solution of common salt set in a cold glass funnel, and each time the filter clogs a new one is substituted for it."

I followed this process, but not so closely, perhaps, as I ought, straining only through the linen, and perhaps having the salt in excess; and got a thick, syrupy liquid, which did not undergo distinct instantaneous coagulation at a lower temperature than 113° F. The frogs I used were frozen almost

stiff, and had been absolutely torpid for months, and to this circumstance I attribute the slight tendency of the myosin to coagulate, so that the proposition may be stated that *long rest of a muscle diminishes the tendency of myosin to coagulate.*

It is very plain *a priori* that these facts must be true of man, if of animals; and clinical observation certainly confirms them.

As a proof of this it is only necessary to refer to a paper on "Instantaneous Rigor as the Accompaniment of Sudden and Violent Death," by Dr. Jno. H. Brinton, in the American Journal of Medical Sciences, 1870, p. 1870. In this exceedingly interesting memoir the sudden occurrence of such instantaneous rigor is very clearly proven by the testimony of the author and other eye-witnesses as being frequently seen on battle-fields. Under no circumstances is the excitement more intense, or the muscular system more strained, than on the field of battle; so that the evidence is certainly complete that both in man and animals excessive exertion predisposes to coagulation of myosin, and renders it liable to alter at a temperature decidedly below the normal point of change.

The cardiac variety of sunstroke is said by the Indian authorities to occur especially in action, rarely during marches, never in barracks. As an example of it may be cited the account given by a witness to Dr. Parkes, and incorporated in his work

on Hygiene (Pract. Hygiene, 2d edit., p. 360), of an occurrence during the first Chinese war. The 98th Regiment was marching on a very hot day, and the surgeon who was with the rear-guard stated to Dr. Parkes that the men fell suddenly on their faces, as though struck with lightning, and, on running up and turning them over, many of them were already dead. Dr. Maclean, who was present at this occurrence, confirms the account given by Dr. Parkes, but states that it was at the attack on Chian-Kiang-Foo. The men were thickly clothed, with tight accoutrements and tight, rigid stocks, and were charging up a very steep hill. A great number of them were stricken down, and fifteen died instantly, falling on their faces, and giving merely a few convulsive gasps.

The points worthy of note in this history are,—first, that the men were making violent exercise going up-hill, so that the heart must have been in exceedingly active exertion, increased by the impediment to the circulation afforded by the tight accoutrements; second, that the attacks were instantaneous and syncopal in their character. To my mind the conclusion is irresistible that death was produced by sudden coagulation of the myosin of the cardiac muscle, and consequent rigid arrest of the heart's action.

Vallin seems to think that the *arrest of respiration*, when it occurs before or simultaneously with

the cessation of the heart-beat, is due to a coagulation of the myosin of the diaphragm. I think, however, that the disproof of this, except as a mere possible rarity, is so plainly contained in what has been said about the heart, that it is not necessary to dwell upon it. I will merely refer to Experiment 10, in which, although the cat died of sudden arrest of respiration, the diaphragm distinctly reacted to the stimulus of galvanism fifteen minutes after death, and also to the fact that is shown in my clinical records, that in man general muscular rigidity is often not developed within the first hour after death from insolation.

The prolonged study of the muscular system in insolation has, then, produced the following conclusions :

1st. Excessive rigidity of heart, due to a coagulation of its myosin, is a very pathognomonic lesion of sunstroke.

2d. That in most cases it is a post-mortem, rather than an ante-mortem, phenomenon, occurring directly after death.

3d. In certain cases, the so-called cardiac variety of sunstroke, death is probably due to a sudden ante-mortem coagulation of the cardiac myosin, and consequent instantaneous arrest of the heart's action.

4th. That the muscles after death from heat fever very soon become rigid, sometimes do so

instantly, and that such rigidity is of the same nature as ordinary post-mortem rigidity.

5th. That while it is conceivably possible that death from asphyxia may occur from coagulation of the myosin of the diaphragm and other respiratory muscles, it is exceedingly probable that in man death never does actually occur from such cause.

Nervous System.—Having completed the study of the action of heat upon the muscles and their pathology, so to speak, in sunstroke, it seems next in order to take up the nervous system. To any one who has witnessed a case of *coup de soleil*, the importance of such investigation must be most obvious. What is required is the determination how far the nervous symptoms are produced directly by the heat, and how far they are the result of antecedent changes in the blood. The first step in such inquiry must logically be the endeavor to find out whether the degree of heat manifested in sunstroke will of itself produce nervous symptoms; and if so, how these will compare with those of *coup de soleil.*

This endeavor also naturally divides itself into two parts: first, the study of the action of heat on the nerve-centres; second, the investigation of its action on the nerve-trunks.

There is no difficulty in applying heat directly to the brain of the cat and rabbit by surrounding

the head with a double bonnet of india-rubber, or, as I have used, of pig's-bladder, and allowing hot water to run through this. Vallin is, so far as I know, the only observer who has made any such experiments. It is evident that there are two points especially to be determined in this inquiry: first, How do the symptoms produced compare with those of ordinary *coup de soleil?* second, What is the temperature at which the functional power of the brain is lost? In only two instances did Vallin succeed in causing death by the hot-water bonnet, and in neither of these cases was any attempt made to measure the temperature of the brain. The symptoms are not described by Vallin as closely as is desirable, but appear to have been insensibility,—whether coming on gradually or suddenly is not stated,—with convulsions.

My own experiments are as follows:

EXPERIMENT 13.—A full-grown rabbit. Rectal temperature 102½°.

Time.	*Temp. of Water.*	*Remarks.*
12.11	140° F.	
12.49	175° F.	Puffiness and great swelling of the scalp, with very hurried respiration and exceedingly rapid pulse, with violent struggles, constitute the only effects as yet produced.
12.51	175° F.	A sudden, severe convulsion, followed by a state of semi-unconsciousness.
1.4	150° F.	Rectal temperature, 104½°.

Time.	*Temp. of Water.*	*Remarks.*
1.10	140° F.	Lies quiet, semi-unconscious; but the corneæ are very sensitive.
1.15	180° F.	Convulsions.
1.20		Died in a stupor, a gradual deepening of the previous semi-unconsciousness. The respiration ceased before heart's action.

Autopsy.—Skull opened instantly, just sufficiently to allow a thermometer to be plunged in the brain; it indicated 117° F. The heart was soft and flaccid; the right side full of blood, the left empty. The muscles responded well to galvanic stimulus, but rigor mortis set in in a few minutes.

EXPERIMENT 14.

Time.	*Temp. of Water.*	*Anal Temp.*	*Remarks.*
12.38	190° F.		
12.50	140° F.	103¾° F.	
1.05	135° F.		
1.15		104¼° F.	Pupils not contracted.
1.20	174° F.		
1.30			Before this there have been struggles, apparently semi-convulsive, and contracted pupils. Now a true convulsion, followed by unconsciousness and complete relaxation. The breathing is accompanied by fine, sonorous râles.
1.50	150° F.	106° F.	The rabbit has lain for some time in a perfectly comatose state, with occasional convulsions. The hot-water bonnet was now removed from the head, and cold water poured over the latter; almost immediately the ani-

mal showed signs of recovering, and after awhile did so perfectly. The next day, excepting in regard to the local trouble in the scalp, etc., the rabbit seemed well.

EXPERIMENT 15.—A young, half-grown cat. The hot-water bonnet was adjusted to its head, and the water allowed to run through it.

Time.	*Temp. of Water.*	*Remarks.*
11.15	162° F.	
11.25		Cat has had several convulsions not preceded by signs of nervous disturbance, coming on suddenly and followed by insensibility, with partial anæsthesia of cornea.
11.40	140° F.	
11.41	162° F.	
11.55	170° F.	There have been repeated convulsions, during which pupils would dilate some, although the eye was in the full blaze of sunlight. Almost constant convulsive trembling, affecting very markedly even the eye-muscles. The cat is all the time absolutely unconscious. Cat died at 11.57, the respiration ceasing at least four minutes before the heart ceased to beat. On opening the body the right heart was gorged with blood, and, on being cut, the ventricle pulsated again. The brain was opened as soon as possible after death. Its temperature was 113° F. The muscles responded to galvanism not so actively apparently as might be, and rigor mortis came on in about fifteen minutes (not timed with watch) after death.

EXPERIMENT 16.—A very large, powerful tomcat was used, and bladder fitted to head at 10.30 A.M.

Time.	*Temp. of Water.*	*Remarks.*
10.35	175° F.	
11.	140° F.	Very hurried breathing.
11.30	180° F.	
12.	160° F.	Cat is now unconscious; the unconsciousness came on in a very short space of time, but not with absolute abruptness; no convulsions; respiration slower.
12.15	175° F.	Cat just dead. The respiration certainly ceased before the heart some seconds, or probably a minute or two. No convulsion.

Autopsy.—Brain opened instantly. The thermometer plunged directly into the substance rose above 114° F.; when placed, however, so as to be in contact with the inside of the skull, it marked 115°. There was decided, but not extraordinary, congestion of the brain.

As these experiments are painful ones, I have not repeated them further. They seem sufficient to establish the following conclusions: *First.* A temperature of the brain of from 113° to 117° F. is sufficient, if maintained, to produce death in a short space of time in mammals by arrest of the respiration. *Second.* That the chief symptoms induced are insensibility and convulsions, preceded by exceedingly rapid respirations and action of the heart, and unaccompanied by any general rise of temperature. *Third.* That these symptoms come

on very quickly in all cases, at times with absolute abruptness.*

A point to be borne in mind in comparing these temperatures with those fatal to the muscle, is that the death is produced by paralysis of the respiratory centres, and that these centres being at the base of the brain are farthest from the heat, hence it is very probable they may have been one or more degrees below the temperature of the cerebrum as represented by the degree noted. The resemblance of these symptoms, induced by the local application of heat to the head, to the nervous phenomena of sunstroke, is very striking, both in regard to the symptoms themselves and also to the suddenness of their onset. A reference to the account of the exposure of cats to a general high heat, will show that in these animals the nervous symptoms are much more sudden and severe than in rabbits; in fact, approaching what is seen in man. A study of the experiments just detailed will show that a temperature of 113° to 114° F. is fatal to the brain of the cat, 117° F. to that of the rabbit.

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* These experiments also incidentally confirm the modern belief that sunstroke is not due so much to the direct heating of the head by the sun, as to the heating of the general body, for the symptoms induced outside of those arising directly from the cerebrum were not those of true insolation. The importance of these experiments in the interpretation of the nervous symptoms of fever, it would be out of place here to dwell on.

The nervous system of the cat is much more excitable, and much more impressible, than that of the rabbit, and consequently feels the abnormal temperature more acutely. The brain of a man is much more highly organized, and no doubt correspondingly more sensitive, than that of a cat; and if a temperature below 113° F. is fatal to the brain of a cat, whose normal temperature is 102½° F., it seems very certain that the temperature of some cases of insolation (113° F.) is sufficient in itself to cause death in man, whose normal temperature is 99° F.

As will be shown hereafter, death in *coup de soleil* is frequently due in a great measure to a slow, gradual deterioration of nerve, muscular, and hæmic organization; and in such cases the fatal result may be brought about without being immediately caused by the temperature, without, in other words, the lethal nerve heat point being reached.

Nerve-Trunks.—According to Vallin, E. Harless published some years since in Henle und Pfeuffer's Zeitschrift, 3d B. viii. pp. 122–185, a paper entitled "Ueber den Einfluss der Temperaturen und ihrer Schwankungen auf die motorischen Nerven." I have not had access to this memoir, but Vallin states that no study of the effect of heat upon nerve-centres was made, and that the following

results were arrived at in regard to the nerve-trunks. If a motor nerve of a frog be gradually heated up to 30°–32° C. (86°–89⅗° F.), its irritability is progressively increased, but at 35° or 36° C. (95°–96⅘° F.) the excitability is abruptly lost. In mammals this loss of excitability occurs at 52° C. (125⅗° F.). In birds, at 57.5° C. (133½° F.). At precisely the degree at which in the different species of animals the irritability is lost there is also a molecular change in the nerve-substance, the myeline fusing so that the nerve-fibre becomes softer and more transparent, and its diameter greater. If a heated nerve be allowed to cool, it regains its functional power; but on a second or third repetition of the heating, its functional ability is finally totally lost.

My own experiments have not been directed so much to the close physiological study of the action of heat on nerve-trunks as to the answering of the question, Does the highest temperature reached in insolation affect seriously the conducting power of the nerves? They were made by exposing for several inches the sciatic nerve, passing underneath it a strip of india-rubber cloth, so as to isolate it from the muscles, and pouring water of a known temperature over it. At irregular intervals the conducting power of the nerve was tested by touching it with the poles of an induction battery above the heated portion. The animals were kept throughout in a state of complete anæsthesia.

The experiments were as follows:

EXPERIMENT 17.—A young pup.

LEFT NERVE.

Time.	*Temp. of Water.*	*Result.*
½ min.	140° F.	Nerve transmits no impulse.
5 "	None applied.	Has partially recovered its power.

RIGHT NERVE.

Time.	*Temp. of Water.*	*Result.*
6 min.	120° F.	No impairment of activity; in fact, nerve more excitable than normal.
17½ "	120° F.	No impairment of activity.
5 "	140° F.	Nerve fails to transmit the strongest impulse.
5 "	None.	Nerve has not recovered itself.

EXPERIMENT 18.—A young rabbit. Crural nerve employed.

Time.	*Temp. of Water.*	*Remarks.*
1 min.	118° F.	
2 "	116° F.	
2 "	124° F.	
1¼ "	122° F.	Excitability of nerve seems increased.
2 "	134° F.	
2 "	129° F.	
1½ "	140° F.	
3¼ "	134° F.	There is not yet any distinct impairment of conducting power of nerve.

EXPERIMENT 19.—A large cat.

Time.	*Temp. of Water.*	*Remarks.*
8 min.	120° F.	
4 "	130° F.	
5 "	124° F.	
5 "	122° F.	
5 "	127° F.	
3 "	123° F.	No distinct impairment of conducting power of the nerve.

EXPERIMENT 20.—A very weak, sick cat.

LEFT CRURAL NERVE.

Time.	*Temp. of Water.*	*Remarks.*
5 min.	136° F.	The nerve was not tested for five minutes after heat was applied. The conducting power was then good, but somewhat lessened.
3 "	134° F.	Nerve transmits impulse only with difficulty.
5 "	None.	Nerve has partially recovered itself.

RIGHT CRURAL NERVE.

Time.	*Temp. of Water.*	*Remarks.*
6 min.	132° to 134° F.	Some slight impairment of the conducting power of the nerves.

As far as they go, these experiments certainly confirm the statements of Henle and Pfeuffer, and demonstrate that the highest temperature ever reached in sunstroke is not sufficient to sensibly affect the conducting power of the nerves.

Coma.—It is very evident from the experiments which have been detailed, that heat exerts a much more deadly influence upon the nerve-centres than upon the nerve-trunks, and that a temperature not higher than that reached in most cases of sunstroke in man, not only induces symptoms similar in many respects to the nervous phenomena of insolation, but will also cause death by paralysis, from over-stimulation, of the respiratory centres. It becomes, therefore, a matter of grave interest to decide, if possible, what is the lowest degree of

heat which is sufficient to produce coma and convulsions.

At first I was somewhat at a loss to invent an experiment which should settle the point. It is manifestly impossible to have the bulb of a registering thermometer in the brain so as to mark the point at which convulsions come on.

I finally determined, as the best method at my command, to apply the hot-water bonnet to the head of an animal, and immediately after the induction of decided nervous symptoms to open the skull and plunge a thermometer into the brain. When the coma comes on gradually, so that it is impossible to determine the time exactly when it commences, this method is very imperfect, but under favorable circumstances is sufficiently accurate.

The following experiments were performed in this way, and I think the results yielded are sufficiently accurate for practical purposes:

EXPERIMENT 21.—A full-grown cat.

Time.	*Temp. of Water.*	*Remarks.*
12.	130° to 170° F. during the hour.	
1.	140° F.	Cat has been for some time very quiet, evidently semi-comatose; at times arousing herself. Pupils moderately contracted.
1.15	180° F.	Cat was so comatose that an attempt was made to open the head. The first incision was not noticed; but the second aroused her.

Time.	*Temp. of Water.*	*Remarks.*
1.30	180° F.	The pupils have been strongly contracted, and cat quiet and semi-comatose. Suddenly pupils at once dilated widely, and a severe convulsion came on. This was so severe that I think the cat would have died in it. In the midst of the fit, however, the skull was opened and the thermometer plunged into the brain. It indicated 108° F.

EXPERIMENT 22.—A young kitten.

Time.	*Temp. of Water.*	*Remarks.*
1.40	170° F.	
1.55		Kitten has been semi-comatose, with strongly contracted pupils, for some time. Suddenly its pupils dilated, and a general epileptiform convulsion, commencing in the muscles of the jaw, set in. In the midst of this the thermometer was plunged into the brain. It indicated 107½° F.

For reasons before given, it seems irresistibly probable that if a temperature of 108 F. in the brain of a cat will produce these serious symptoms, it will induce no less in man.

The conclusion is apparently unavoidable that the nervous symptoms of sunstroke may be, and probably are, due to the direct action of the heat upon the cerebro-spinal axis, and that death itself by asphyxia is often brought about by the same influence.

The only objection of any force which I can imagine capable of being raised against the conclu-

sion just drawn from the previous experiments, is that the results were not really due to the immediate action of the heat, but to a determination of blood to the head and consequent congestion of the brain.

The want of validity of this objection is apparently demonstrated by the following facts:

1. Sudden epileptiform convulsion is not generally the result of congestion of the brain.

2. Opening the skull through the longitudinal sinus, although necessarily affording immediate relief of any existent congestion, did not stop the convulsion.

3. Abstraction of the heat by the pouring of cold water over the head, sufficed to produce immediate cure.

Dr. Richardson, of London, in a lecture published in *The Medical Times and Gazette*, takes a different view of sunstroke. He says, "There is little difficulty in explaining the cause of the coma of sunstroke: it is due to the contraction of the vessels which supply the brain with blood, and to the subsequent changes in the nervous system incident to withdrawal of blood."

Unfortunately, Dr. Richardson does not deem it necessary to prove this explanation, and it therefore rests merely upon his *ipse dixit*. The experiments herein detailed certainly show that whatever

may be the immediate method through which it acts, high temperature within the brain causes the nervous phenomena, and to invoke the aid of the capillaries in explanation of this fact seems to me to be unnecessary, and, in the absence of proof, gratuitous. I think the more direct and therefore more logical supposition is, that the heat overstimulates the nerve-cells themselves. More than this, what little evidence there is points strongly towards the latter conclusion. It is impossible to believe that during the comatose stage of *coup de soleil* there is a state of general vaso-motor excitement, because in both animals and man the general, if not absolute, rule is, that during the coma the pupils are minutely contracted. This is equally true whether the applied heat be general or restricted to the encephalon.

As a part of the convulsion, there is no doubt often vaso-motor spasm, with sudden, abrupt, wide dilatation of the pupil; but all these appear to be secondary, not primary, phenomena, preceded, not followed, by changes in other nerve-centres than the vaso-motor.

In order to prove that what is the possible and also probable cause of any effect is the absolute cause, it is often necessary to disprove other asserted causes, and therefore the full establishment of the fact that heat is the real primary and immediate cause of the nervous disturbance of sun-

stroke, must be put off until the pathology of the blood is considered.

The Blood.—As has already been stated under the head of Pathology, the blood in sunstroke suffers very markedly. In man it is always black, or very dark, semi-fluid, with its alkalinity impaired,—in a word, presenting largely the appearances and conditions which exist in it in cases of low fevers. In my experiments in animals, it has partaken of these characters to a degree, but not so obviously as it appears to have done in the experience of Claude Bernard and Vallin. Both of these observers, struck by the dark color of the blood, have made researches as to its gases.

The method which Claude Bernard employed was substantially as follows: By means of a syringe, blood was drawn from the vena cava of an animal just dead of sunstroke, and passed without exposure to the air into a glass receiver, previously fitted to a mercury-pump. The gas, which was obtained from the blood by the latter, passed immediately into a graduated measure. It of course consisted of oxygen, nitrogen, and carbonic acid. The last having been absorbed by means of potash, and the first by the use of pyrogallic acid, what was left represented the nitrogen. From 100 cubic centimetres of blood were obtained:

Blood, 100 c. c. { CO_2 37.2 c. centim.
O 1.00 " "

In a second experiment there was a momentary exposure of blood to the air. The result was as follows:

	CO_2	27.9 c. centim.
Blood, 100 c. centim.	O	3.8 " "
	N	9.4 " "

Normal venous blood, treated in the same way, yields, according to Claude Bernard, from 12 to 15 c. cent. of oxygen per 100.

The analyses given by Vallin (p. 150) yielded the following results. Gas obtained in 100 volumes of venous blood:

Normal Blood.	*Heat-Stroke, Exp.* 1.	*Heat-Stroke, Exp.* 2.
CO_2 37.26 c. c.	36.79 c. c.	35.96 c. c.
O 13.81 "	0.94 "	0.75 "
N 2.62 "	3.96 "	1.70 "
53.69 "	41.69 "	36.41 "

After the addition of sulphuric acid to the blood:

Normal Blood.	*Experiment* 1.	*Experiment* 2.
CO_2 9.37 c. c.	6.42 c. c.	1.51 c. c.
O 1.12 "	0.57 "	0.38 "
N 1.25 "	3.21 "	0.38 "
11.74 "	10.20 "	2.27 "

These analyses, having been made by different investigators, and being entirely in unison, and also in accordance with the pointings of all the other facts of the case, must, I believe, be accepted as demonstrating that after death of animals from

heat the proportional amount of oxygen in the blood is only a fractional part of what it normally should be, and that the dark color of the blood is largely owing to this absence of oxygen.

The question which presents itself is, To what is this absence due? That it is not due, as might be at first supposed, to such a destruction of the life of the blood as to prevent the absorption of oxygen, is shown by the fact that if the blood be drawn from the vein during life, or directly after death, and be shaken in the air, it rapidly assumes the arterial tint. Claude Bernard (loc. cit., p. 185) explains poverty in oxygen by stating that when blood has its temperature elevated it has a very great power of changing oxygen into carbonic acid, and that the phenomenon is really a post-mortem result; for if the "body be opened instantly after death, and the arrest of the heart's action, the blood is still red in the arteries, especially in cases where death has been produced simply by the breathing of the hot air, the body of the animal remaining outside of the stove." As to the latter class of cases I cannot speak, having had no experience. The conditions are so artificial, so unlike those of genuine sunstroke, that it has not seemed necessary to make the experiment. I have, however, examined a number of cases of heat-stroke in animals, and in all of them the blood was everywhere dark, even when the animal was killed before the appearance

of any indications of immediate death. To me the explanation of the small quantity of oxygen in the blood in sunstroke, both before and after death, is very simple. Oxidation under the stimulus of the high temperature must go on with intense rapidity; and, no doubt as a direct result of this, the respiration very early in the affection becomes immensely accelerated, and for a time this rapidity serves to maintain more or less fully the balance of demand and supply. But by-and-by the respiration begins to fail; although no less rapid, it grows shallower and less perfect, and the constantly increasing demand is met by a constantly diminishing supply, until, in many cases, death results from gradual asphyxia, and long before life is extinct, the livid, purple surface, the dark, stagnant capillaries, indicate what is taking place.

The fact that the red globules are generally able to absorb oxygen after death, would indicate that the blood has not lost its vitality: that it is not dead.

This indication receives very strong corroboration from the experiments of Max Schultze (Schultze's Archiv, i.), who found that when blood was heated in a moist chamber, under the microscope, no alteration took place in the red globules until a temperature of nearly 52° C. ($125\frac{3}{5}$° F.) was reached, when they became contracted and broke up into débris and little masses of coloring matter

At a temperature of from 45° to 46° C. (113°–114⅘ F.) the amœboid movements of the white corpuscles became more active, and continued so for a greater or less length of time. When the temperature was raised to 50° C. (122° F.) they stiffened, as it were, retaining permanently the condition or shape in which the heat found them.

Eulenberg and Vohl, according to Vallin, have advanced the theory that death from sunstroke is the result of the sudden liberation of gases in the blood. According to both Hermann (Virchow's Archives, t. lxii.) and Vallin (Archives Générales, Jan. 1872), the work of these authors is a most remarkable mixture of all that is bad in scientific treatises. Vallin gives at some length the reasons of Eulenberg and Vohl, and, as their proofs do not amount to anything whatever, and the whole theory is very unreasonable, I am content to simply refer my readers to the reviews by Hermann and Vallin.

A more plausible theory is one originally proposed by Weikard and recently supported by Dr. Richardson, of London. (St. Bartholomew Reports, vol. vii.) I have not seen the paper of the former of these authorities, but according to Vallin (Archives Générales, Jan. 1872), the memoir appeared in the "Archiv der Heilkunde," 1863, p. 193, under the title of "Versuche über das Maximum der Wärme in Krankheiten." In this paper Weikard states that one of the dangers of high

temperature is an augmentation of the coagulability of the blood and consequent formation of clots in the vessels. As quoted by Vallin, the principal proof brought forward is that the coagulation of blood occurs more rapidly outside of the body when it is heated than when cold. The following table expresses one of his experiments:

		1	2	3
Man, 20 to 30 years old. Apoplexy.	Commencement of coagulation,	T. + 23° 1 minute.	T. + 43° Instantly.	T. + 23° 1 minute.
	Firm coagulum,	20 "	12 minutes.	18 "
	Separation of serum,	12 "	8 "	10 "
	Diameter of clot after 2 hours,	34 cent.	33 cent.	?

The result reached by Weikard was, that by a temperature of from 36° to 40° C. ($96\frac{4}{5}$°–104°F.) the coagulation is retarded, below and above that it is accelerated.

Dr. Richardson, of London, in the lecture in *The Medical Times* previously alluded to, expresses a similar belief. He says, "At this stage there may be slower motion, gradually falling temperature, and collapse. These changes indicate in nearly every instance that there has been separation of fibrine in the heart. The separation has stopped or arrested the current of the blood at the main, and virtually all is over. In the human subject we recognize by external signs this condition con-

stantly; in the inferior animals we produce it synthetically, and determine it with precision.

"Here, in illustration, is the dead body of a cat. In this animal there was, during life, an induced increment of heat or fever. The fever progressed until the mean temperature of the animal had reached an increment of nearly 10 degrees; then the animal began to sink. She might have struggled on hopelessly for hours, as human bodies do; but we could in her case put her to death, in sleep, by making her inhale the vapor of ether. We did so, and at once laid bare the heart. See what has happened. The right cavities are almost filled with a firm separation of fibrine."

Coagulation of the blood in the heart is certainly not a common occurrence in animals killed with heat. I have examined a large number of such hearts and have never seen any coagula. I have also examined all of the arteries of some cases, without finding thrombi. As will be shown hereafter, if a pigeon be revived after having been nearly killed by heat, it recovers consciousness, but remains partially paralyzed, and finally dies. It occurred to me that possibly this might be due to coagulation of the blood in the small capillaries, and, to test the matter further, the following experiments were instituted:

EXPERIMENT 23.—Two pigeons were killed by the hot box. Two hours afterwards their brains were carefully examined

with the microscope. The capillaries were almost all of them empty; a few contained masses of blood-corpuscles, none of them, however, firm clots.

EXPERIMENT 24.—Pigeon was put in hot box, and when dying, its head was cut off. In the examination of the brain capillaries I failed to detect any thrombi.

EXPERIMENT 25.—A pigeon was partially revived from heat-stroke, and its head then cut off. A careful microscopic study of the brain gave the same negative results as in the other cases.

As the results of my experiments, I am forced to refuse assent to the idea that coagulation of the blood in the living body is a factor in the history of sunstroke, and to explain the single experimental result deduced by Richardson as due to the slow death of anæsthesia. Clinically viewed, the matter appears even more definite. Loss of coagulability of the blood is the one great lesion of human *coup de soleil*, to whose existence all observers bear witness; and in the absence of all evidence of real weight, it seems most illogical to conclude that death is ever due to the formation of coagula during life.

In my experiments upon animals, I have never witnessed the profound breaking down of the blood often seen in human sunstroke. Other experimenters have had different experience; and, after all, the difference is one of degree, not of kind. The absence of these blood changes in many of the cases, especially in the most rapid and when the nervous symptoms are the most severe, shows that they are

secondary, not primary, in their position. Indeed, the whole testimony of the facts which have been elicited in regard to sunstroke is in accord with this view, for in rapid cases there is, as already stated, comparatively little deterioration of the blood. In slow cases, the vital fluid suffers the same degradation that is seen in other fevers with high temperature. When once these changes have been set up, it cannot be doubted that they react upon the nervous system, and possibly at times serve to keep up the disease. In human sunstroke, running a course mayhap of many hours or even days, the secretions are largely arrested, whilst tissue changes are vastly accelerated; more effete matter than normal is thus poured into the blood, and the avenues of discharge are choked up; so that both directly and indirectly the vital fluid is degraded, until at last it in some cases no doubt becomes incapable of sustaining life.

Before making a general review of the subject, it remains to establish the proposition already given,—that heat is not merely a possible and probable, but the absolute and only cause of the primary nervous symptoms of sunstroke.

It is not requisite, after what has been said, to go much into detail, for it is an almost absolutely necessary conclusion from what has been demonstrated, that the changes in the blood are not the

cause of *coup de soleil.* It is, however, comfortable to have this conclusion experimentally verified.

It has been shown that the life of the blood is not destroyed, and that there are no embolic arrests of circulation. There remains only the possible, but exceedingly improbable, supposition, that a specific poison is developed in the blood. Dr. Stiles (loc. cit.) has proven that the injection of the blood of an animal dead of heat-stroke into the veins of a second animal, is not productive of harm to the latter. Surely I am justified in believing that it has been shown that all possible causes (except heat) of the primary nervous disturbance have no real existence as such, and in asserting the truth of the proposition that abnormal temperature is the only poison present.

If this be so, withdrawal of the heat before too much damage has been done, ought to be followed by immediate relief of the symptoms. The following experiments show that this is the case:

EXPERIMENT 26.—A young rabbit was put in a glass box set in the sun; in twenty minutes he was apparently totally unconscious, having passed through all the ordinary symptoms. He was now taken out, and put in a bucket of water. The temperature of his body rapidly fell to normal, that of the water rising two degrees, and consciousness was restored at once. He was very weak, but in a few minutes was able to walk some, and the next day was as well as ever.

EXPERIMENT 27.—A pigeon of full age, with anal temperature 108½°, was placed in hot box (130° F.) at 11.42 A.M. At

12 M. it had a convulsion, followed by persistent opisthotonos, with complete unconsciousness, which indeed had been nearly complete before the convulsion. 12.2 P.M. the pigeon was taken out, utterly unconscious, and at one time I thought it was dead: there were only a few gasps at long intervals. I plunged it into a tub of cold water, and kept it there. Its respirations slowly improved; but after it had been in some three or four minutes it had a violent convulsion, after which for awhile it again appeared to be dead. It, however, slowly got better again, and in about fifteen minutes was taken out of the water and put in the air. It was now perfectly conscious, and breathing slowly and regularly, but was not able to walk. In two or three minutes it was able to push itself rapidly forward with its feet, on its breast, but was unable to raise its body from the ground. Its anal temperature was 100°. It was now left in a basket at 1 P.M., apparently improving. At 2 P.M. it was found dead, still warm. I saw the body at 4 P.M. There was general rigidity, and the blood was not coagulated anywhere.

EXPERIMENT 28.

Time.	*Temp. of box.*	*Anal Temp.*	*Remarks.*
11.40	120° F.	105° F.	
11.48	120° F.	112° F.	
11.55	120° F.	117° F.	The pigeon had previously struggled violently, but its struggles were apparently voluntary. It was so weak as not to be able to walk, and was now taken out of the box. It was unable to stand at all. 12.8 P.M. the anal temperature 112½°; pigeon is now able to push itself along, although not to stand. 12.30 P.M. pigeon apparently all right, but not disposed to fly, and its feathers seem ruffled. Pigeon died some time between 2 and 5 P.M. Autopsy made twenty-four hours afterwards showed that the blood was fluid, and very dark.

EXPERIMENT 29.—Adult pigeon. Anal temperature, 109½°. At 12.29 P.M. it was put in hot air chamber. 12.39 P.M., anal temperature, 112°. 12.53 P.M., anal temperature, 117½°. Bird lying on back and side, apparently dying. It was plunged in cold water for several minutes, and when taken out its anal temperature was 109½°. It was unable to make any effort; with very irregular, jerking breathing, so that I momentarily expected it to die. It was not again put in water.

2 minutes (after taking out of water).—Temperature, 107°.

3 minutes.—General condition growing worse.

10 minutes.—Reviving. Able to push itself along.

12 minutes.—Temperature, 101½°. On application of galvanic current, muscles respond well.

25 minutes.—Although pigeon has been in a warm place, its temperature is 96°.

2 hours.—Pigeon much better; lies quiet all the time, but can walk, though constantly falling; still, is better. It has been dry and in a warm place for two hours, but its temperature is only 100°.

3 hours.—Left as before.

5 hours.—Found dead, cold and rigid. Blood as first taken out dark and fluid, but on standing in a test-tube forming into a firm coagulum.

These experiments certainly demonstrated what has been claimed. The after-death of the pigeons is probably explainable as the result of permanent damages done to the nervous system, due to the intense heat.

Theory of Disease.—Sufficient progress has now been made in this memoir to render necessary a short review of the facts that have been developed, preparatory to a final sketch of the nature of *coup*

de soleil. It will be called to mind by my readers, no doubt, that it has been shown that the sole efficient, always present, cause of sunstroke is excess of temperature; that whilst circumstances of the degree of moisture in the air, or of the condition of the patient, as race, acclimatization, temperance, health, and so forth, have great influence, yet they possess such influence only in so far that they render the heat more penetrating or efficient, on the one hand, or the patient less capable of resisting the heat by the natural methods of self-cooling, on the other; that the disease is generally preceded by more or less marked prodromes, and that when it is fully developed the symptoms consist simply of intense fever, with great nervous disturbance, as shown by unconsciousness, paralysis, convulsions, etc., and that in most cases death results from gradual asphyxia or a simultaneous failure of circulation and respiration; that there are cases whose course is almost like that of lightning-stroke,—instant death,—the patient suddenly falling forward in a condition of fatal syncope, and that these cases always occur during active exercise; that the most obvious lesions found after death are, a condition of the blood similar to that seen in low fevers, and great rigidity of the heart.

In the second or experimental portion of the memoir it was shown,—

That sunstroke may be produced in animals as

readily as in man, either by natural or artificial heat; that the symptoms are similar to those seen in man; that death takes place ordinarily by asphyxia; that after death the characteristic lesions are alteration of the blood and rigidity of the heart, with immediate or quickly appearing post-mortem rigidity of the general muscular system; that this rigidity of the heart comes on in most cases after, not before, death, and is a result, not cause, of death; that post-mortem rigidity is dependent upon coagulation of myosin, and that the rigidity of the heart is of similar origin, coagulation of the muscle plasma occurring almost instantaneously at 115° F., a degree almost attained in sunstroke; that when a muscle has been in great activity immediately before death, the myosin coagulates at a much lower temperature, and that the cases of sudden cardiac death occurring in battle among the East Indian English troops were no doubt due to the coagulation of the heart's myosin; that heating the brain of a mammal produces sudden insensibility, with or without convulsions, at a temperature of 108° F., and death when a temperature of 113° F. is reached; that this effect of the local application of heat is not due to induced congestion, but is the result of the direct action of the heat upon the cerebrum, and that consequently the nerve-centres are as perniciously affected by high temperature as the muscles are;

that the nerve-trunks bear a temperature of 125° F. without their conducting power being immediately affected; that whilst the general symptoms induced by heating the brain of a rabbit are very different from those of sunstroke, the nervous symptoms are exactly similar; that the life of the blood is not destroyed by any temperature reached in sunstroke, the amœboid movements of the white blood cells and the absorption power of the red disks not being injured; that the amount of oxygen of the blood is greatly lessened, as the result of gradual asphyxia combined with abnormal consumption of oxygen; that there is no reason for believing that capillary thrombi are common in sunstroke; that there is no specific poison developed in the blood; that the deterioration of the vital fluid is due to the rapid tissue changes induced by the fever, and the more or less complete arrest of excretion; that such deterioration is secondary to the nervous symptoms, not primary; that if the heat be withdrawn before it has produced permanent injury to the nervous system, blood, or other tissues, the convulsions and unconsciousness are immediately relieved, and the animal recovers.

As a postulate from these facts and deductions, I think it follows that the *nature of sunstroke is that of a fever*, or, in other words, that *coup de soleil is a fever, not dependent upon blood-poisoning, but upon heat,—an ephemeral or irritative fever*, if the term be preferred.

It is well known that certain lesions of the nerve-centres will cause fever without the supervention of inflammation or blood-poisoning; such a lesion is section of the spinal cord in the neck. Indeed, it is apparently inconceivable that inflammation itself can cause fever in any other way than by a reflex action upon the nervous system. Further, every practitioner has seen fever, and even "the fevers," produced or developed largely through the nervous system. Very recently I have had a patient convalescent from severe typhoid fever, in whom the temperature for a week had not been over 99° F. She, contrary to orders, got out of bed, and made considerable exertion, and within fifteen minutes had a slight rigor, followed by intense fever, the temperature rising at once to 104°, and in a few hours to 107° F. At the same time, grave nervous symptoms were developed, and the pulse rose above 150, so rapid and thready that it could not be counted. There are two explanations of these facts, either of which is conformable to our present knowledge:

First. That fever depends simply upon paralysis of the vaso-motor nerves.

Second. That there is in the medulla or higher up a controlling centre, which influences through the vaso-motor or other nerves the heat production in the body.

Which of these be the truth it is not necessary

for our purpose to be determined, the theory of sunstroke being plain in either case. When a man is exposed to heat beyond his powers of resistance, there is a gradual slow rise of temperature, until the stimulus of the heat becomes so intense as to paralyze either the heat centre or the vaso-motor nerves, as the case may be, and then there is probably a sudden intensifying of the oxidation processes, and a further rise in temperature, preceded, accompanied, or followed by an overwhelming of the cerebrum; in other words, intense fever, with its accompaniments, is developed.

That sunstroke is of the nature of a fever was dimly seen by Sir J. Randall Martin and other Indian writers, but not distinctly postulated by them, at least that I know of, and the standard authorities of England to-day do not consider sunstroke among the fevers.

It is said that Dr. Gerhard formerly taught in his clinical lectures that sunstroke is a fever; but I am not aware that he has put such opinion on record.

In a paper in the American Journal of the Medical Sciences, Jan. 1859, Dr. J. J. Levick makes an elaborate comparison between the post-mortem results of typhus fever and sunstroke, and says, "These facts would seem to indicate that there is in sunstroke, as there is in typhus fever, a poisoned condition of the blood, and that it is to this that we

are to refer the various morbid phenomena of the disease. I cannot, however, divest my mind of the belief that in sunstroke this unnatural condition of the blood is a secondary affection, not the primary one; that there is, in the first place, an exhaustion or depression of the nerve-forces which regulate nutrition, respiration, circulation, and the other acts of organic life."

In 1863 I published a paper in the American Journal of the Medical Sciences, in which I insisted strongly upon the claim of this disease for a place among the fevers, and gave it a name as such, namely, *Thermic Fever*. I believed at that time that the nervous symptoms were due to the development of a poison in the blood, not perceiving that the heat itself was the poison.

In the last edition of his Practice of Medicine, Dr. George B. Wood allowed the claim of sunstroke for a place among the fevers. He says, "Heat is a universal stimulant to the organs; and excessive stimulation, or rather the high degree of irritation that attends it, has a tendency first to derange, and in the end to diminish or suspend, the functions of these organs." In accordance with these views, Dr. George B. Wood proposed the name of *Heat Fever* for the disease. Since the difference between the two names seems to be very slight, I have retained Thermic Fever, as having the priority.

PART III.

TREATMENT.

The treatment of sunstroke has been very various in the past, just as the ideas as to the nature of the affection have been so diverse. By the first observers it was looked upon as the result of rush of blood to the head, and the quick, often full and hard pulse, the hot skin, suffused face and eyes, the sudden unconsciousness, the convulsive startings, all seemed to them to call most imperatively for bleeding. Accordingly, they practiced venesection, and for a time believed they derived advantage from it; but the frightful mortality which attended this practice soon forced the abandonment of the lancet, and latterly venesection appears to be condemned as a general practice both by American and Indian surgeons, the testimony of some of whom I subjoin.

Dr. Dowler, of New Orleans, has occasionally seen bleeding apparently do good, yet, as a general measure, he unqualifiedly condemns it. He says (New York Medical Gazette, 1842), "During the

last five years I have been called to see a very considerable number of sunstruck persons, within five or ten minutes after they fell in the streets. Formerly, I used to bleed them, and though the great heat of the body is thereby suddenly diminished, the pulse becoming soft as air, yet by the time the arm is tied up (which is done for form more than for necessity), the patient is suddenly choked, and, to appearance, by a dense tenacious mucus, the breathing not ceasing gradually, as in other diseases, but instantly, the face turning livid, and even its veins, especially upon the forehead, becoming at the moment distended. Bleeding hastens the strangulation, though it is always desired by friends."

Dr. Swift (New York Medical Journal, 1854) condemns bleeding, and also the indiscriminate use of affusions. Looking on the cases as instances of cold collapse, he advises that the patient be put in a hot bath, rendered stimulant by capsicum or mustard, and that general stimulation be used.

Surgeon Thos. Longmore, of the Indian army (London Lancet, 1859), condemns venesection, except in rare cases, and extols cold affusions, with purgative enemata, and mustard-plasters along the spine.

Dr. Wm. Pirrie, of the same service, says, "I have been told by many who had had ample means of observation, that venesection always

seemed to hasten a fatal termination." He commends cold affusions to head and chest, with diligent use of frictions and heat to legs and arms.

Crawford says (The Madras Quarterly, Oct. 1860, p. 330), "I have never seen any advantages derived from venesection in such cases, and I am satisfied that the reverse is often the case. It is worthy of remark that its strongest advocates admit 'that blood-letting kills, if it does not cure the disease,' and that 'to deplete with good requires care and discrimination, aided by a calm judgment.'"

Dr. G. S. Beatson (Med. Times and Gazette, Dec. 1857) condemns bleeding very strongly. He states that, in an epidemic which he witnessed, four cases were treated by venesection, with three deaths, whilst of eight cases treated by repeated cold affusions to head, chest, and epigastrium, all recovered.

Dr. A. E. L. Longhurst (Lancet, Jan. 1869) commends treatment by pouring cold water freely on the head, and the internal use of croton oil in small quantity, with the free administration of brandy. He gives three cases, all of which recovered under this treatment. He believes the cold water to act by exciting the nervous system, and states that in his cases the immediate effects were raising of the pulse and forcible contractions of the voluntary muscles; but if the douche were continued too long at one time, it was followed by diminution of

the volume of the pulse and marked depression. He condemns venesection.

Mr. J. R. Taylor, Deputy-Inspector of Hospitals, India, says, after describing a severe epidemic of the disease, "Had I fortunately before the epidemic at Gozupore, 1843, met with the records to which I have referred, I should have been more moderate in the employment of venesection." He then speaks of an epidemic which he witnessed in 1852, in which "numerous" severe cases were treated simply by continuous cold affusions without one death.

Further testimony might be adduced as to the impropriety of bleeding in sunstroke; yet it cannot be gainsaid that occasionally whilst the blood has been flowing the patient has become conscious. Dr. Dowler appears to admit this freely, although condemning venesection. The truth appears to be that whilst, in some cases, blood-letting may do good, in by far the majority of instances it does absolute harm. There can be no doubt of the great refrigerating power of bleeding. Dr. Dowler expressly states that the skin grows cool when it is practiced in *coup de soleil.** I conceive that when

* Since this memoir has gone to press, on re-reading Dr. Wm. Pepper's account of sunstroke (referred to in the early part of this work), I find that all his cases had been freely bled before he saw them. The cases were very probably true sunstroke, not simply heat-exhaustion, as I have suggested, and the cool skin was the

venesection does good, it does so generally, if not always, by reducing the temperature; and since other simpler and infinitely less exhaustive means of doing the same thing are so available, bleeding is generally exceedingly bad practice, even if the patient does recover.

In order, however, to allow the other side of the question to appear somewhat, I append the following from a report in the *Medical Times and Gazette*, October, 1870:

CASE XI.—Dr. J. H. Salter was called to a lad 16 years of age, who before the attack had been in perfect health. Dr. Salter, on arriving half an hour after the lad fell, found him "insensible, and wriggling in convulsions like an eel."

He says, "The muscles of the trunk, neck, and legs were rigid, the countenance suffused, the eye directed backwards and fixed, the breathing snatchy and stertorous.

"On closer examination, I discovered the pupils were contracted to a minimum, the conjunctivæ were injected, and the teeth were firmly clinched together. The temperature of the body was abnormally high, with the surface dry and rough. The heart beat tumultuously, and the throbbing of the carotid was very energetic.

"At the wrist the character of the pulse was small, quick, sharp, and wiry; there was a lack of volume, not of propelling force. I watched him carefully for some minutes, and his symptoms increased in severity, so that I considered him in extremis. I then took several ounces of blood from

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result of the bleeding. The soft, flaccid hearts were found because, the temperature not being high at the time of death, the cardiac myosin did not coagulate.

his arm without delay. The effect was most striking. The convulsions were quickly relieved, and gradually ceased as the blood continued to flow; the pulse became slower and fuller, the breathing less labored and snatchy, and the venous congestion of the face subsided; the pupils relaxed a little, and by the time I had bound up his arm he lay in a heavy, oppressed sleep.

"He was then removed to a neighboring house and put into bed. Cold applications were used to his head, and his extremities, which were now hot, were artificially warmed. A dose of castor oil was given as soon as he could swallow, and through the night small quantities of weak beef-tea and brandy.

"The next day the patient was much better and partially conscious. The following day he was able to be taken home."

In remarking on the case, Dr. Salter says, "The immediate effect seemed to be to relieve the general venous congestion; to render the breathing calmer and less labored; to diminish the frequency and alter the character of the pulse; to lessen the spasms and arrest the convulsions."

The Indian treatment of sunstroke appears to be almost uniform of recent years, having the cold douche for its basis. Barclay, Aiken, Maclean, Morehead, Avary, and others, might be cited, but further quotation seems scarcely necessary, and would be cumbersome. At the same time, whilst experience has taught the Indian surgeons the value of the cold douche, it appears evident that most of them consider that it acts chiefly by rousing

up the nerve-centres: a false theory, which must impair in practice the efficiency of the employment of the remedy.

In America, no such uniformity of treatment has as yet prevailed, although in the last five years the drift of the professional teaching seems to be more in that direction.

In 1859 (American Journal of the Medical Sciences, vol. xxxvii. p. 55), Dr. B. Darrach recommended that ice be applied to the skin, and also rubbings with ice, in sunstroke; and this treatment has subsequently been employed quite largely and been generally approved, especially by Dr. Levick (Pennsylv. Hospital Reports, 1868).

In the same Reports for 1869, Dr. Jas. H. Hutchinson details a number of cases in which hypodermic injections of morphia were employed. Sixteen were so treated, and all recovered but four: a result which certainly is much better than my own experience in the same hospital. At the same time, it is to be noted that in most or many of these cases the "ice treatment" was more or less efficiently employed, so that the results cannot fairly be attributed solely to the morphia.

It will be noted that I have avoided making any statistical comparisons of results of treatment. I have done this from the belief that such inquiry would be worse than useless. The condition of the patient before the attack, the severity and form

of the latter, and the time at which treatment is begun, are so rarely noted, and are so diverse and so important in affecting the results, that all statistical comparisons seem futile.

In concluding this essay, the final, practical question is, What is the best method of treating sunstroke? And I shall now attempt the answer of this.

If there be any truth in the views of the nature of thermic fever taken in this memoir, it is self-evident that the chief indication in every case is the abstraction of heat. An excess of the latter agent is the *materies morbi;* and if it can be removed before too much harm has been wrought, the patient will assuredly recover.

As soon, therefore, as a man falls, he should be carried into the shade with the least possible delay, his clothing removed, and cold affusions over the chest and body practiced. This must not be done timidly or grudgingly, but most freely. In many cases the best resort will be the neighboring pump. In the hospital, probably, a better method of reducing temperature than the cold affusion is to place the patient in the cold bath (50° F.), or rubbings with ice may be practiced, and, as originally suggested by Dr. Parkes, enemata of ice-water may be given.

In using these various measures, it must always be borne in mind that the indication is the reduc-

tion of temperature: if the means employed do not accomplish this, they do no good.

Relaxation of the pupil is said to be "the first symptom that shows the good of the cold affusion" (Aitken, Practice of Medicine, vol. ii. p. 394); but as, in my experience, the pupil frequently has not been contracted, too much reliance cannot be placed upon this; and I think the thermometer in the axilla, or better the rectum, affords the best guide as to the effect of the treatment.

After the temperature has been reduced, and even after consciousness has returned, there is often a great tendency to a fresh rise of temperature and consequent relapse. This tendency may be met by wrappings in wet sheets, and, if necessary, by a recourse to the more powerful measures for reduction of temperature, which have been already cited.

It must be borne in mind, however, that the cold douche, cold bathing, etc. are powerful remedies, and are capable, if used too long, of doing harm.

In my experiments upon animals, I have seen the temperature, when once reduced by them after sunstroke, continue, notwithstanding their withdrawal, to fall until it was many degrees below normal. It needs no argument to establish the harmfulness of such reduction, and, whilst the possibility of its occurrence should not intimidate us in

the use of the measures, it should temper our boldness with caution.

The evidence adduced by Dr. Hutchinson in favor of the hypodermic use of morphia, as first suggested by Dr. Herbert Norris, is certainly sufficient to justify its further trial.

Dr. Hutchinson has stated to me, in conversation, that the effects of the remedy were most apparent in the convulsive form of the affection, and that, indeed, the power which it exhibited in allaying the spasms was most surprising.

Moreover, theory seems rather to favor than disfavor the practice, as it has been shown by Prof. Binz that morphia has the power of checking tissue changes, and thereby lowering the temperature. It seems impossible that the moderate use of morphia can do harm: it may do great good; and I would, therefore, advise the hypodermic injection of a quarter of a grain of the sulphate every four to six hours, especially in the convulsive cases.

After the first violence of the attack has been subdued, a blister may be applied, as recommended by some English authorities. Aitken says (loc. cit., p. 394), "The hair is to be cut short so soon as possible, and a blister applied to the nape of the neck, the surface having previously been first well sponged with *acetum lyttæ:* . . . increasing confidence as to the ultimate result may be indulged in so soon as vesication takes place; and, in cases

where insensibility recurs, after an interval of ten or twelve hours it may be removed by the application of a second blister to the vertex."

While I would personally use a blister in this way, from deference to authority and in the hope it might do good, I would not rely too much upon it.

Many of the Indian authorities advise a mild purgative; in most of the cases that I have seen, there was already diarrhœa; a dose of Rochelle salt would, however, do no harm, but in bad cases could hardly be swallowed early enough to do much good.

Both Drs. Barclay and Maclean speak of a class of cases in which every attempt to administer the cold douche produces such violent convulsions that it is inadmissible. It seems probable that the cold baths, or rubbings with ice, would be borne by some of these cases; if so, they no doubt would be of inestimable value.

The authorities alluded to have never tried these measures, but have used chloroform by inhalation.

Dr. Maclean says, "In India I have saved the lives of many by acting on this advice" (*i.e.* giving chloroform). Dr. Barclay, in like manner, found chloroform useful "in the convulsive form of the disease attended with extreme nervous irritability: . . . in some instances life was saved by the remedy; in all it was prolonged."

As already stated, it is especially in these cases that Dr. Hutchinson has found the hypodermic use of morphia of service. The best treatment of the convulsive form of the affection would, therefore, seem to consist in the use of morphia and chloroform, with the cold bath, or ice rubbings; the latter measures being practiced, if necessary, whilst the patient is in a state of anæsthesia.

PART IV.

SEQUELÆ.

It* is *a priori* very improbable that an affection which produces so rude a shock to the nervous system as does thermic fever, should not frequently be followed by grave after-effects.

Experience confirms such probabilities, for in many cases, after recovery from the acute disease, various symptoms of deranged innervation manifest themselves.

Comparatively little has been written upon the sequelæ of the affection, but among Indian authorities, Maclean states that the least serious of these after-effects is "incapacity for service in India, or any hot climate, without at least a more or less prolonged stay in a cold climate." This inability to endure heat in some degree almost universally

* This portion of the monograph has been added since the Boylston prize was awarded.

follows sunstroke. It generally, with care, wears off, sometimes in a year or so, sometimes not till after a period of years.

Persistent headache, very severe and very intractable, is spoken of by Maclean as a more serious sequela.

Whilst serving in our army hospitals during the rebellion, I saw quite a number of men who had been disabled from duty by *coup de soleil*, and this cephalalgia, often associated with giddiness, was a very common, almost universal, symptom.

Unfortunately, I kept no notes of cases; but, through the kindness of Dr. Harrison Allen, I am allowed to publish the following brief notes, taken by him during his service. From the fact that the hospitals he had charge of were well towards the front, but few of such chronic cases remained for any length of time under his care, so that complete clinical histories were not obtainable; but the abstracts given are sufficient to show the general symptoms.

Case XII.—Wm. L. received a sunstroke May 6, 1864, during the battle of the Wilderness. Lay unconscious one hour and a half. On recovery, had severe headache, and has suffered from it very much since. He had, previous to the attack, convergent strabismus of left eye, which has been much worse since. He also lost the sight of the eye at the time of the accident. Jan. 1865, he had his first convulsion; has had three altogether.

Tongue at present furred; pulse small, thready; sleeps

well; bowels not costive. He suffers from persistent frontal headache.

CASE XIII.—David L. received a sunstroke in July, 1862, which was followed by drowsiness, headache, and vertigo. He was ill for several months. Up to present time, July, 1863, has not been able to do duty.

At present time is anæmic, with a calm pulse of 72, a clean tongue, slight epigastric tenderness, and some dyspeptic symptoms; bowels regular. There is loss of memory, and at intervals there are attacks of vertigo and weakness, lasting several days.

CASE XIV.—George W. Sunstroke one year ago. Before this time the patient had never had headache. Since the occurrence has had it constantly. There is marked loss of memory. Bowels constipated. Has attacks of vertigo and weakness on taking even gentle exercise. Exposure to sun produces intense headache and vertigo. He sleeps but very poorly. Mouth temperature 99° F. Pulse 95.

CASE XV.—B. L., about a year since, during a march after the battle of Gettysburg, fell senseless, and remained unconscious one hour. Since this he has suffered from constant intense headache and obstinate constipation. The bowels are never moved except by the aid of medicine. He sleeps poorly, and suffers from decided loss of memory. Exposure to the sun increases the headache very greatly. Reading causes dizziness and great pain in the eyes. Pulse 95. Exercise produces great palpitation of the heart.

Intense, fixed headache, with or without other serious indications of brain lesions, is often the most prominent symptom in these cases. The fixedness and obstinacy of the pain would seem to indicate

that it is more than functional, an indication confirmed by the frequency with which other and more serious symptoms of brain-trouble are finally manifested. Thus, Dr. Pepper, in the paper previously quoted, states that out of twenty cases treated in the Pennsylvania Hospital, three of the ten recoveries resulted in chronic brain diseases, eventuating in insanity.

In order to treat these cases intelligently, it is of very grave importance to decide what is the real nature of the trouble.

Some time since, Dr. S. Weir Mitchell expressed, in conversation, the belief that in many, if not all, of the serious cases the after-lesion of thermic fever is *meningitis;* and reflection has convinced me of the truth of his position.

There can be no doubt that in many cases of sunstroke, after the subsidence of the original disease, there is manifested a marked tendency towards the development of inflammation of the meninges of the brain. This has been noticed by several observers: thus, Bonnyman (Edinb. Medical Journal, 1864) speaks of reaction sometimes setting in strongly, and requiring the application of leeches to the temples.

Some years since, Dr. Mitchell, after walking about the city for nearly the whole of an intensely hot August day in the sun, went out of town a few

miles, and climbed a steep hill in the intense blaze of the afternoon sun. At this time his carotids were throbbing tremendously, and he had an intense, bursting headache. He sat down to dinner a few moments after this, and shortly fell forward upon the table, entirely unconscious.

He says that he has a dim remembrance of being helped up-stairs; but beyond this is an absolute blank, in his memory, of ten days. During this period he is said to have had an intense fever, with delirious unconsciousness, and a rapid, feeble pulse.

He was treated with cold applications, and at the end of the period named, after a prolonged mustard foot-bath, recovered his consciousness.

The first sensation was that of an intense, agonizing headache, and the next was the perception of his father—Dr. J. K. Mitchell—and Dr. Mütter discussing the possibility of his recovery.

As soon as he could speak, he said, "Bleed me." His father, coming to the bed, shook his head; but the son repeated, "Bleed me." He persisted in simply repeating this, until they thought him delirious; but at last he mustered strength to say, "I am not delirious, but have a frightful headache: if you don't bleed me I will die." By this time his pulse had become full and bounding, and finally he sat up and was bled. Ten or fifteen ounces were taken without avail; but as more blood flowed the headache vanished, to be succeeded by a feeling of

most delightful languor and rest from pain. Between twenty-five and thirty ounces were taken, and afterwards recovery was a very simple matter.

That Dr. Mitchell, by the bleeding, was saved from meningitis appears most probable, especially in the light of the following case, reported by Dr. J. J. Levick in the Transactions of the College of Physicians of Philadelphia:

CASE XVI.—A lad, æt. 17, had been engaged during the morning in unloading coal at a wharf in New York. While doing so, he complained of pain in the head, and fell over in a convulsion. He remained in a state of stupor, and was brought to Trenton in the canal-boat, unprotected, and thence by railway to Philadelphia.

At the time of his admission he was in a semi-stupid state, but could be roused with some exertion. He had at that time ptosis of the right eye, with occasional delirium and other symptoms of inflammation of the brain, for which latter affection he was put upon the use of calomel, in small and repeated doses, blisters to the nape of the neck, etc. After death there was found extensive inflammation at the base of the brain.

Dr. S. Weir Mitchell has generously allowed me to publish the following cases, which have occurred in his practice. They are strongly corroborative of the opinion already advanced as to the nature of the sequelæ of sunstroke.

CASE XVII.—G. B., æt. about 35, ordinary seaman; previously well; no syphilis. Being in the United States navy,

he was exposed to prolonged sun-heat on the African coast, while on some duty connected with overseeing a party of natives who were watering the ship. He came on board suffering from headache, which grew worse in the hot tropic night. The next day he fell on the deck, and soon became insensible. He thinks he was treated with purgatives, and cold to the head, but can recall little of what passed during three weeks, when he became well enough to be on deck. He was told that he had had sunstroke, and not the coast fever. Save rare headaches, he had no permanent evil results, but the least exposure to sun made him suffer with parietal pain and with weakness. Two years later, in Norfolk, a long exposure to heat produced general feebleness and headache, which lasted a month or more. Thenceforward his headaches were frequent and severe in summer, but less common and more mild in cold weather. He entered my wards at St. Joseph's Hospital two years after his last stroke, and had then great general weakness and constant headache, with buzzing in the left ear. As well as I remember, the senses were otherwise unaffected, and the lungs, heart, and kidneys healthy. There was, however, slight acid dyspepsia, and intense constipation. A great variety of treatment had failed to relieve his pain, which was severe, was worst during the day, and was only eased by heavy doses of opiates. A few days after he entered, he became sluggish and inattentive. This was followed by lessened complaints of headache, and somewhat later by stiffness in the muscles at the back of the neck, with contracted pupils, coma, and death. On examination we found a remarkably extensive meningitis, some of it old, and some new. The later trouble lay chiefly at the base, and there was no other brain lesion proper.

The following case seen very recently offers many points of likeness to that just related:

CASE XVIII.—Mr. ——, United States army, a sturdy, well-made man, æt. 30, married four years, but childless, was in perfect health until August, 1869, when serving in Texas he contracted ague, which proved very persistent. While still liable to rare attacks of this disease, he was called upon to ride a long distance in a very hot sun. On his return he dismounted, and, while standing by his tent, he began to feel headache, very suddenly. In addition to this he had true vertigo, external objects turning around him, and violent throbbing of the head, especially behind his ears, and also in the arteries of the neck. His face was deeply flushed, and I presume from his statement that he must have felt just such symptoms as follow a full inhalation of nitrite of amyl. He staggered into his tent, and lay down, feeling weak and confused. An illness of six weeks ensued, with headache, delirium, much worse at night, and high fever. On recovering it was thought unwise to keep him on duty, and he was most imprudently sent back on horseback to San Antonio,—a four days' ride. A relapse occurred, and he was again ill, with like symptoms, during two weeks, when he gradually recovered, the headache becoming by degrees more rare, until in the summer of 1870, after exposure to sun-heat, the cephalalgia became again more frequent and more severe. The winter which followed alone gave full relief. In April, 1871, in New Mexico, the great heat renewed his pain, and before July he fell twice, owing to sudden weakness with vertigo. During this summer he had frequent headaches, with many dizzy spells, which at length were accompanied with loss of consciousness without spasm. At this time also he suffered from sense of choking, and there was also steady pain high up in the right side of the neck, with buzzing in the right ear, tenderness of the mastoid process, and stiffness of the muscles below and back of this region. The attacks lessened with cooler weather, and in November and December there were none. I should

add that from July he had had, when excited, tremor of the right arm, but, notwithstanding these grave symptoms, had remained on duty. January 1, 1872, when much annoyed and angry, a sudden spell of giddiness came on, with numbness of the right arm, and twitching of the right hand. From thence forward he dragged the leg a little in walking. He came to Philadelphia in March, 1872, and in the interval had several attacks, which were alike, and began with a sudden increase of the post-aural pain, which he properly says is a sense of numbness with pain. The pain seems to him to ascend above and behind the ear, and to run forward on to the forehead. Meanwhile, the head feels full, vertigo comes on, objects seem to rotate from left to right, and loss of consciousness, without spasm, follows, after which he sleeps, or is dull and drowsy. Until he came to this city the leg and arm were increasingly feeble, the tremor easily excited, and the headaches frequent. The palsy was not, however, always the same, and in a time of excitement and danger, during his homeward journey, he suddenly seized the reins of a pair of unruly horses, and then discovered that for some days after he could walk with ease. Perhaps, feeling his weakness, he had not before this ventured so much, and this seeming return of power was only a discovery of a capacity always existing.

Mr. —— had no new attack until June 1, 1872, at which date he was free of headaches, and was rapidly gaining strength. The first attack in June was preceded by pain down the right neck, shoulder, and arm. A second fit followed, June 9, and it then appeared that all his symptoms had darkened.

When seen by me in consultation, June 12, 1872, this was his condition: The mind said to be unimpaired; memory unaltered, or, if at all changed, it is so in regard to names; temper as usual; questioned closely, he thinks that an hour before an attack his memory of dates and names becomes

much obscured; hearing unaffected, but at times has had buzzing in the right ear; taste and smell as usual. I detected no lesion of either eye bearing on the case. After the attacks he has seen double for an hour, but now there is no squint, no difficulties as to the accommodation. The field of vision is entire, the acuteness of vision normal, and the pupils, alike in size, play readily under changes of light. Dr. Wm. Thomson was kind enough to see Mr. —— with me, and he also found no changes in the disks of either eye, and no evidence in them of past or present mischief. The sensibility of the neck and face was examined with care, and with these curious results: The entire distribution of the fifth nerve was dysæsthetic, while also over the eye and in front of the ear the sense of touch was lost altogether. The ear and cheek and lips were least affected, but there was still more complete loss of touch over the whole back of the neck and head to the middle line. A needle-prick was felt anywhere in the face, but not at all below the ear, or at any point of the neck from the third cervical vertebra to the occipital bos.

The mastoid process feels at times painful, and is slightly tender on pressure. Deep pressure on the muscles below and behind the right ear causes some pain, and he is never free from some sensation in this region, which ranges from annoyance to intense pain. The state of tactile sensation in the right hand varies, and has varied so as that at times he has slight loss of feeling, at others much more. When first examined by me, the localizing power and the sense of pain and temperature were all lessened. The compass-points could not be separated widely enough to be felt as two on the fingers of the right hand. The feeling of the right leg was rather less disturbed.

There were no motor-palsies of any of the cerebral nerves. The muscles of the right side of the neck were said to be at times stiff, and were always sore. Speech normal. The

right hand had, by the dynamometer, one-quarter the power of the left. The right hand was in a state of tremor, increased by the examination. During perfect rest it did shake. When he made voluntary motions with the hand it ceased to shake. The limb had every movement, but each was feeble. He could walk with only a slight shuffle in the gait.

Heart, lungs, abdominal organs, healthy; appetite good; digestion natural; bowels regular; urine, two specimens, specific gravity 1024, no deposit, no albumen or sugar.

The treatment consisted of iodide and bromide of potassium, with corrosive sublimate. The post-aural region was leeched four times. When the first leeches were applied he did not feel them, but before they fell off he felt them. The second leeching was felt at once, and the effects were manifest in the almost entire return of feeling in the fifth nerve and its partial restoration in the cervico-occipital region. The pain also disappeared, and the head felt lighter and better. From thence the gain in other respects has been steady and remarkable.

June 29, 1872.—The leeching was repeated yesterday, and three ounces taken. A previous dry-cupping afforded less relief; that from the leeching is notable, and what is thus gained is also kept. He thinks the sensation of the hand better, but it is still so dull that the width of the fingers is not space enough to enable me to separate the compass-points so that they shall seem to be two. On the neck and face the sense of touch is still improving, and everywhere a light contact is felt, save at the centre of the cheek, over a space a half-inch in diameter; even here a deep pressure or rough touch is felt. Mr. —— ordered to the seaside, and to continue treatment.

This case is one of general interest. There was no suspicion of specific disease. It was clearly due to sunstroke. In all probability there was some inflammatory state of the

membranes surrounding the upper end of the cervical cord, and a like state of the basal cerebral membranes. Both centres of disturbance must have been on the right side. The diagnosis, although not altogether satisfactory, seems to accord pretty well with the results of treatment, which have been decided and satisfactory. Many of his symptoms might lead one to suspect the presence of a tumor, but I do not see how a mass could be so situated as to occasion all the troubles, while the careful and repeated studies of the eye leave no doubt as to the absence of the usual ocular signs of basal tumor.

I think the evidence now brought forward is sufficient to establish the frequency of meningitis following sunstroke, and that the fixed, intense headache so often complained of is probably due to such cause.

Whether the epilepsy and insanity which occasionally follow sunstroke are really due to chronic meningitis or not, I cannot say.

Maclean states that the epileptic fits usually subside when the patient is removed to a cool climate.

There is often, however, after *coup de soleil*, a condition of simply deranged innervation, with headache, dyspepsia, etc., in which there is no sufficient reason to believe that actual inflammation of the meninges exists. Such cases are readily distinguished by the headache being irregular and shifting, sometimes absent, sometimes present, sometimes frontal, sometimes occipital, etc.

Treatment.—If the view here taken be correct, the indications for treatment are very plain. First and most important among them is the avoidance of exposure to heat, it being evident that every renewal of the original irritation will add fresh fuel to the fire. This being borne in mind, the treatment resolves itself into that of chronic meningitis, which it is not necessary here to discuss. It is, however, interesting to note that at Netley, England, without apparently any definite idea as to the true pathology, they have, as the result of clinical observation, settled upon a good plan of treatment; for Maclean (*loc. cit.*) says, "When the pain is fixed and severe, long-continued counter-irritation to the nape and a course of iodide of potassium sometimes permanently relieve."

In the milder forms of the after-affection, the physician's duties may often be limited to simple hygienic measures and treatment of symptoms.

In these cases, as well as in the more serious, Dr. Maclean lays stress upon the necessity of attending to the functions of the skin, which he states always to be profoundly perverted.

THE END.

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